THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—25TH YEAR. SYDNEY, SATURDAY, JUNE 25, 1938. No. 26.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

Cerebral Vascular Accidents and their Treatment, by C. T. CH. DE CRESPIGNY 1077
The Surgical Treatment of Essential Hypertension: Early Results in Three Cases, by GILBERT PHILLIPS, M.S., M.Sc. 1081
The Treatment of Head Injuries in General Practice, by R. A. MONEY 1084
Some Aspects of the Leptospirosis Problem in Australia, by W. C. SAWERS 1089

REPORTS OF CASES—

Regional Enteritis, by DAVID B. SKEWES, M.B., B.S. 1097
A Case of the Adult Form of Still's Disease, or the Still-Chauffard Syndrome, by C. G. LAMBIE 1098

REVIEWS—

Biology for Medical Students 1100
"The Compleat Pediatrician" 1100

LEADING ARTICLES—

Babel 1101

CURRENT COMMENT—

Alkalis and Pleonexy 1102
The Administration of Thyreoid Gland to Premature Babies 1103

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 1104
Nominations and Elections 1108

CORRESPONDENCE— Page.

The Copper Sulphate Treatment of Tropical Ulcer and New Guinea Mouth Disease 1108
Physical Therapy in Oto-Laryngology 1109
National Health Insurance 1109
Migraine from the Allergic Viewpoint 1111

OBITUARY—

Leslie Wylie Norman Gibson 1112

MEDICAL PRIZES—

The Stawell Memorial Clinical Prize 1112
Norman Gamble Fund and Research Prize 1113

UNIVERSITY INTELLIGENCE—

The University of Sydney 1113

THE BRITISH ASSOCIATION OF RADIOLOGISTS—

Thesis for Fellowship 1113

HONOURS—

Birthday Honours 1113

BOOKS RECEIVED 1113

DIARY FOR THE MONTH 1114

MEDICAL APPOINTMENTS 1114

MEDICAL APPOINTMENTS VACANT, ETC. 1114

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 1114

EDITORIAL NOTICES 1114

CEREBRAL VASCULAR ACCIDENTS AND THEIR TREATMENT.[1]

By C. T. CH. DE CRESPIGNY,

Consulting Physician to the Adelaide Hospital.
Adelaide.

I PROPOSE to discuss these vascular accidents according to their situation, considering first hæmorrhage within the meninges and then passing to intracerebral vascular lesions of sudden onset.

Subdural Hæmorrhage.

Subdural hæmorrhage is also known as chronic subdural hæmatoma or *pachymeningitis hæmorrhagica*. When I was a student in pathology, Sir Harry Allen, in the course of his lectures, used to show specimens of *pachymeningitis hæmorrhagica*. He pointed out that this condition was seldom recognized during life, but was not infrequently found *post mortem* in very old subjects, in chronic alcoholics and in the incurably insane. These people had died after a period of stupor followed by coma.

During the last twenty years our knowledge of the clinical manifestation of chronic subdural hæmatoma has become more precise and its mode of production more accurately known through the classical article by Wilfred Trotter.(1) The immediate cause of chronic subdural hæmatoma is trauma. The patient receives a blow upon the head, usually caused by a fall; most often it is at the back, but sometimes it is in front. Consciousness is not lost and the impact is seldom sufficient to cause concussion. Naturally, in the case of a person of unsound mind or chronically addicted to alcohol, the fall may be forgotten; and the same is true of an individual whose memory is impaired by age.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 31, 1938.

As the result of the blow the brain is shaken like a jelly on a dish, and some of the delicate vascular channels between the soft meninges, clothing the lips of the great longitudinal fissure of the cerebrum, and the longitudinal sinus, are torn. Slow leaking of the blood takes place, usually on one side only, but sometimes on both. There seems to be no natural tendency for the bleeding to stop. Clotting takes place in layers, and after a time, owing to the breakdown and liquefaction of the central part of the coagulum, a blood cyst is formed. The central part is fluid, usually dark brown, but sometimes of a chestnut colour. It contains changed hæmoglobin and hæmatoidin crystals. The wall of the hæmatoma is laminated blood clot. The mass occupies the space between the dura and the arachnoid, extending forwards, downwards and backwards to a variable extent. The cerebral hemisphere is compressed and its ventricular cavity is obliterated. The whole process lasts for several weeks, and this accounts for the march of its symptoms. There is usually a latent period of two or three weeks after the fall or bump on the head, and it must be remembered that the blow or fall may have been quite slight. In some cases, even in mentally alert people, the trauma may have been forgotten.

The first symptom is usually intractable headache, though in some cases remissions of the pain may occur. The headache may be more severe at the vertex or may be general. The mental state in the early stage is unaltered. After some days, change of manner, temporary aphasia and forgetfulness may be noted, and the patient may appear cheerful in spite of pain, and may seem to be lacking in insight. At this time there may be no local neurological signs, or slight inequality of the pupils may be noted. Such inequality seems to be of slight value in detecting the site of the lesion. Some days or weeks later the patient is somnolent, but can be roused. At this stage it has been noted in some cases that the breathing may be of a curious heavy character; and even while awake the patient's respirations resemble those of a sleeping person. Now there may be signs of slight hemiplegia, such as slight facial weakness, diminished or absent abdominal reflexes and perhaps Babinski's reflex, with increased deep reflexes on the same side. These signs may, however, be absent throughout, or Babinski's sign may be bilateral. Slight diplopia is sometimes present. Ocular paralyses or pareses may also occur at this period, if not before.

Lumbar puncture shows the cerebro-spinal fluid to be under a low pressure. It may contain a few red blood cells and slight excess of protein, or it may not depart from the normal.

As time goes on, the sleepiness becomes stupor and later coma, leading to death. Respiration may be slow or cerebral in type towards the end, and may finally fail before the heart ceases to beat. In this later stage the patient becomes incontinent of urine and fæces.

Such is the history of a so-called typical case, but there are many in which the symptoms are much less suggestive. They may occur in a different order; for example, diplopia of a transient character may occur early, or the patient may be stuporose or comatose when first seen, and no reliable history may be forthcoming.

Other cases are described, in which even coma and incontinence have completely disappeared, with apparent recovery, and later the patient has relapsed and died. Such variation depends on the rate of compression, on the capacity of the brain to adapt itself and on the extent and site of the hæmorrhagic process.

Cases have also occurred in which the victims were young adults without a history of trauma, in whom the blood cyst has been found in the middle fossa and at the base.

If treatment is undertaken before the patient is in the last stage, recovery may take place. In deeply stuporose or comatose patients the compression and ischæmia of the brain have usually led to irreversible changes.

Case Reports.

CASE I.—A woman, aged about fifty years, fell on the deck of a steamer whilst disembarking. She cut her left eyebrow. She was able to pick herself up. Three days later she had severe headache, which continued. On the ninth day she was drowsy, but still complained of headache. When examined twelve days after the fall she was drowsy. There was slight paresis of the left side of the face and of the left arm and lower limb. Slight left ptosis with slight dilatation of the left pupil and slight external strabismus were present.

Dr. L. Lindon operated. Right subtemporal craniotomy revealed no abnormality. The left side was then opened and a subdural black clot, the size of the palm of the hand, was evacuated. The brain had been compressed and very slowly expanded. The patient made an uninterrupted recovery.

CASE II.—On December 10, 1937, a man, aged fifty-two years, was struck on the right eyebrow by the hames on the harness of a restive horse. The brow was swollen and bruised afterwards.

I am indebted to his medical adviser, Dr. Cyril Piper, for the careful record of his history after the accident.

The patient seems to have been slightly stunned by the fall. Then he suffered from a constant frontal headache of variable intensity. He was usually sleepy. He always felt weak, and when he tried to read he became more tired and had to stop. On one occasion he saw double for a while. After a week or so his personality altered. From being rather reticent by nature he became garrulous and somewhat vapid in his conversation. Gradually his expression became heavy and dazed.

I examined him ten weeks after the accident and found him exactly as Dr. Piper had described. His eyes and cranial nerves were normal. The left finger-to-nose test was slightly fumbling. The deep reflexes were present in the arms, but were hard to elicit in the lower limbs. The plantar reflexes were normal. The patient was slightly shambling in his gait. Romberg's sign was absent.

The pressure of the cerebro-spinal fluid revealed by lumbar puncture in the horizontal posture was 100 millimetres of water, rising rapidly to 170 millimetres on jugular compression. The examination of the fluid revealed slightly increased protein, but no other abnormality. Skiagrams of the skull revealed no abnormality.

Dr. L. Lindon performed encephalography. The air did not enter the ventricles and the convolution pattern was not easy to define.

Twelve weeks after the accident the patient began to be incontinent of urine during sleep.

On March 24 Dr. Lindon opened the cranium in the left parietal region and removed a mass of flat clot containing dark brown fluid. Three days later a similar procedure was carried out on the opposite side. The patient, on March 31, was already improved. By the end of May, 1938, he had returned to work.

Treatment.

The correct treatment as soon as diagnosis is made is to trephine the vertex near the middle line. This can be done under local anæsthesia. If the clot is present the *dura mater* looks tense and purple. The clot may be removed, or if it is very extensive the fluid contents may be drained out slowly to avoid reactionary cerebral œdema. It is probably wise to open both sides in every case, owing to the possibility of bilateral extravasation.

Subarachnoid Hæmorrhage.

Subarachnoid hæmorrhage occurs in two groups of cases: (*a*) In young people, from congenital aneurysm[2] (occasionally it is familial); (*b*) in elderly subjects with high blood pressure, from rupture of a sclerotic cerebral artery. In severity the condition varies from slight oozing, with correspondingly mild symptoms, to a hæmorrhage profuse enough to cause death within a few minutes or a few hours.

Although in the majority of cases the lesion may be diagnosed from the characteristic march of symptoms, it often presents much difficulty because of unusual manifestations.

I shall describe what may be regarded as a typical moderately severe case ending in recovery.

CASE III.—A lady, aged fifty-six years, was travelling in a sleeping car. In the night she was awakened suddenly by intense pain in the head extending down the back of the neck. At the same time she was nauseated and vomited. Within a few minutes she was stuporose and in an hour or so almost comatose.

When examined she was almost comatose and her breathing was stertorous. There was slight weakness of the left side of the face and the left plantar reflex was extensor in type. During the next three days consciousness gradually returned, but the patient was slightly aphasic and her memory for recent events was absent. About eight or nine days from the onset she had three convulsions within twenty-four hours and her condition became worse. She was stuporose for about twenty-four hours after the fits. Gradually improvement set in and she progressed favourably. For many weeks her mental state was that of Korsakoff's syndrome, but ultimately this disappeared and she finally recovered and was able to lead a normal life again. A very slight weakness of the left side of the face persisted. Lumbar puncture six days after the initial attack revealed yellow cerebro-spinal fluid.

In some young adults a history of headaches resembling migraine, recurring for many years prior to the rupture, may be obtained.

Recovery without residual paralysis may be complete. In some cases residual palsy remains in the form of slight facial weakness on one side, or more extensive hemiplegic signs remain. Some degree of paralysis of the third or sixth nerve on the side of the hæmorrhage may cause a permanent strabismus. Third nerve paralysis, partial or complete, with slight hemiplegic weakness on the opposite side, may persist. Recurrence of the bleeding at a variable time after the initial attack is not infrequent. The second hæmorrhage may be fatal or may result in complete recovery, or it may be followed by the residua already described. In a few cases the extravasated blood collects as a cake of clot in the basal cisterns, leading to internal hydrocephalus, with choked disk and other signs of increased intracranial pressure, which may be the final cause of death.

The important points in diagnosis are as follows: The onset of headache is sudden, the pain extending down the back of the neck, and is quickly followed by nausea. Consciousness may be rapidly lost or impaired or, more rarely, not affected. In hypertietics examination of the eye grounds may reveal arteriosclerosis. In young people this is not seen; but at any age circular hyaline retinal hæmorrhages may appear, usually on the side of the lesion, though they may be bilateral.

The cerebro-spinal fluid during a day or so after the onset is blood-stained to a variable degree, according to the amount of hæmorrhage. In a few days after the hæmorrhage, on centrifugation a pellet of red cells is seen at the bottom of the test tube, with a supernatant layer of yellow but translucent fluid.

Treatment.

Lumbar puncture should be performed for diagnosis, for the relief of headache and for the reduction of pressure. The headache persists for many days after the onset, and usually requires morphine. In cases of recurrent hæmorrhage the question of tying the ipsilateral internal carotid artery must be considered, especially when arteriography reveals an aneurysm.

Intracerebral Lesions.

Vascular leakage and occlusion are the commonest forms of intracerebral lesions.

Cerebral Hæmorrhage.

The cause and diagnosis of cerebral hæmorrhage are too well known to be dwelt upon in a discussion such as this. Unless the hæmorrhage is a very rapid copious outpouring of blood the patient may not become instantly unconscious. I was once the witness of an apoplectic seizure.

CASE IV.—The patient was a nurse, aged fifty years, whose systolic blood pressure was 260 and diastolic blood pressure 140 millimetres of mercury. She had been complaining of headaches and dizziness for some months, and had been admitted to hospital for rest and clinical investigation. Whilst I was measuring her blood pressure one day she suddenly exclaimed "Oh, my head", and then "The side of my face feels funny". She lost consciousness in about thirty seconds and presented the characteristic appearance of apoplexy with stertorous breathing. The left limbs were limp and the left cheek was puffed out with each noisy expiration. The pulse was small and thready. She died about six hours later.

It is doubtful if more than a very small proportion of patients recover from cerebral hæmorrhage.[3] The differential diagnosis is from arterial occlusion and other causes of sudden loss of consciousness with paralysis. Treatment is probably of little avail and should be aimed at lessening the risk of insufflation pneumonia.

CASE V.—A *nullipara*, aged thirty-five years, five months pregnant, felt "headachy" and sick one morning, but nevertheless prepared the midday meal. She suddenly fell unconscious and was admitted to hospital a few hours later, where she died shortly afterwards. Her blood pressure on admission was high and the urine was loaded with albumin. At autopsy an extensive hæmorrhage was found in the right cerebral hemisphere. The liver showed the characteristic subcapsular hæmorrhages and intralobular focal necrosis of eclamptic poisoning. The cerebral vessels were not degenerated, but the ruptured vessel was not identified. In this case it is probable that increased blood pressure and the vascular spoiling by the eclamptic poison were the exciting and predisposing causes of the hæmorrhage.

In purpura (primary or secondary), cerebral hæmorrhage may be the immediate cause of death. This occurred in a patient I saw some time ago with Dr. Clayton.

CASE VI.—The patient, a man, aged fifty-six years, suffered for some six days from *purpura hæmorrhagica*. Death occurred on the eighth day, after sudden loss of consciousness with hemiplegia.

Scurvy, leuchæmia, the enteric fevers and hæmorrhagic forms of the exanthemata are rare causes of cerebral hæmorrhage. Hæmorrhage into or contiguous to a cerebral tumour may cause sudden death or sudden exacerbation of symptoms.

Cerebral Thrombosis.

Arteriosclerosis.—Arteriosclerotic patients with high blood pressure form the majority of victims of cerebral thrombosis. The seizures vary from slight sudden facial weakness or monoplegia, with associated mental confusion and perhaps nominal aphasia, to sudden loss of consciousness with hemiplegic signs. After a while consciousness may slowly return, with a varying degree of aphasia and paralysis as a residuum. During the early stage the urine may contain both albumin and sugar, or either, although the patient is neither nephritic nor diabetic. The eye grounds may show all stages of arteriosclerotic changes, and are to be examined in every case. The blood pressure does not tend to fall to any great extent during the initial seizure.

In treatment, posture is important, and the airway should be unimpeded. "Cardiazol" or "Coramine" may be given. A saturated solution of magnesium sulphate may be injected into the rectum to counteract, if possible, the reactionary cerebral œdema which surrounds the infarcted area in the brain. Morphine should be avoided unless restlessness is extreme. Deep and protracted coma, with unequal pupils, irregular respiration, moist sounds at the pulmonary bases, rising pulse rate and increasing temperature, are bad prognostic signs. The degree of residual paralysis is variable. Subsequent attacks are the rule, but a second seizure may not occur for years, or it may never occur.

Low Blood Pressure.—People with a low or average blood pressure, but having degenerated arteries, form a smaller group of victims of cerebral thrombosis. In these the thrombosis tends to occur in smaller vessels. The attacks are more numerous, but slighter; the distribution is as a rule cortical, and the ultimate result is mental enfeeblement and senile dementia, with or without hemiplegia.

Syphilis.—Syphilis of the cerebral vessels causes *endarteritis obliterans*, with consequent occlusion of the lumen by clotting.

These cases of cerebral thrombosis are indistinguishable in their symptoms and signs from those conditions of thrombosis already considered. Diagnosis must rest upon the absence of other predisposing causes (for example, high blood pressure, age and arterial thickening); it depends often on the comparative youth of the patient, on the history of nocturnal headache, on the presence of other signs of syphilis of the nervous system, pupils, reflexes *et cetera*, and finally on the characteristic changes in the blood and cerebro-spinal fluid. The history of infection and any tertiary syphilitic signs are of obvious importance.

Immediate and energetic treatment should be instituted. Mercurial inunction, bismuth injections and massive doses of potassium iodide should be given, followed by the use of arsenicals.

Such means, however, seldom render a positive Wassermann reaction negative, and a certain amount of paralysis and perhaps some mental enfeeblement and change of character may persist.

The vascular changes of syphilis usually manifest themselves five years or more after the infection. Before the introduction of the Wassermann test we were taught to regard all vascular cerebral disorders in a person under the age of forty years as probably syphilitic if no other cause could be found. It is a rule to be remembered, so as to be kept always on guard.

Apoplectiform seizures in general paralysis, or acute œdema in the neighbourhood of a cerebral tumour, may be pitfalls in diagnosis, although if these conditions are borne in mind a correct conclusion may be arrived at by a careful anamnesis and clinical examination, together with the appropriate serological and other tests.

Cerebral Embolism.

In almost all cases cerebral embolism has its origin in vegetations thrown off from the heart valves or in the mobilization of clots which have formed in the left side of the heart.

Bacterial Endocarditis.—Sudden hemiplegic or monoplegic paralysis is frequent in bacterial endocarditis. Usually seen in rather advanced cases, it may, however, be an early symptom.

CASE VII.—I have recently had under my care a boy, aged nineteen years, whose first recorded symptom was the sudden and temporary appearance of partial nominal aphasia and right-sided facial weakness, together with slight mental confusion. When he was first seen, about three weeks after this occurrence, his speech had become normal, but there was slight facial asymmetry and a small embolic patch was present in his left retina. The course of his disease was subsequently quite characteristic.

Chronic Valvular Disease.—In chronic valvular disease cases of mitral stenosis are those most

frequently associated with cerebral embolism. This occurs sometimes after the use of digitalis or quinidine. The augmented action of the heart dislodges the embolus.

Coronary Occlusion.—After coronary occlusion with myocardial infarction an adherent intramural clot may become detached and cause cerebral embolism.

Aortic Aneurysm.—Aortic aneurysm occasionally causes cerebral embolism.

Other causes exist, but these are the most frequent and should be borne constantly in mind. The ultimate result of embolism in regard to subsequent paralysis is rather more favourable than in thrombosis, though the fate of the patient may depend on the causal condition.

Treatment.—The treatment is symptomatic. Favourable results have been reported to occur in retinal embolism from the use of acetylcholine, but I have not heard of such benefit being derived from the use of this drug in cerebral embolism, nor do I know of any pharmacological experiments to show its action on the cerebral arteries.

The after-care and the general management of resultant paralysis are beyond the scope of this paper.

Temporary Vascular Seizures.

CASE VIII.—Some years ago I had under my care a man, aged sixty years, whose arteries were apparently normal; his systolic blood pressure was 140 and his diastolic blood pressure was 90 millimetres of mercury. He suffered during several years from attacks of temporary aphasia, mental confusion and partial monoplegia. Any individual limb on either side might be affected. The attacks lasted for a few hours and then passed off. They were never severe.

This malady was described very fully by Russell in 1909,[4] and it is a rare but well-recognized condition. The cause is obscure, but it is assumed by most observers to be an intermittent closure of the arteries of the brain, leading to ischæmia of the appropriate area of supply.

Such variable and transient paralyses are sometimes met with in hyperpietics, and it is probable also that the aphasia, hemianopia and monoplegia, which are sometimes associated with migraine, are due to abnormal temporary spasm of cerebral vessels.

Hypertensive Cerebral Attacks.

In people with high blood pressure, especially in those with a diastolic reading above 120 millimetres of mercury, hypertensive cerebral attacks may occur. These are variable in kind and severity, but resemble each other in their usually temporary nature. They have been ascribed to cerebral œdema, and in them the cerebro-spinal fluid is found to be under an unduly high pressure.

The attacks may be epileptiform, with local or general convulsions. A variable degree of monoplegia or hemiplegia may occur. The patient may become rapidly comatose. The blood pressure is high and the retinal arteries are sclerotic. The urine may be normal, or in nephritic patients the specific gravity may be low, and albumin and casts may be present.

Treatment.—Rapid bleeding, up to a pint or more, is good treatment. Castor oil in a dose of 30 cubic centimetres (one ounce) should be given. The bowel should be emptied by an enema. Ninety cubic centimetres (three ounces) of a saturated aqueous solution of magnesium sulphate should be left in the rectum. Lumbar puncture frequently gives rapid relief.

Uræmia.

In the sthenic cerebral form of uræmia, temporary paralysis, muscular twitching and convulsive seizures are probably due to disorders of the cerebral circulation. Purging, sweating and lumbar puncture are often beneficial.

References.

(1) W. Trotter: "Chronic Subdural Hæmorrhage of Traumatic Origin, and its Relation to Pachymeningitis Hæmorrhagica Interna", *The British Journal of Surgery*, Volume II, Number 6, 1914, page 271.
(2) The Bible: II Kings, Chapter iv, verse 18.
(3) J. Collier: "Price's Text Book of Medicine", Fourth Edition, page 1543.
(4) W. Russell: "Intermittent Closing of Cerebral Arteries", *The British Medical Journal*, October 9, 1909, page 1109.

THE SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION: EARLY RESULTS IN THREE CASES.

By GILBERT PHILLIPS, M.S., M.Sc.,
Honorary Neurological Surgeon, Lewisham Hospital; Honorary Assistant Neuro-Surgeon, Royal Prince Alfred Hospital, Sydney.

(From the Department of Surgery, University of Sydney.)

IN a recent article[1] the present writer discussed briefly the indications for surgical treatment in essential hypertension and referred to the results which had been obtained in a group of cases in America. It was pointed out that essential hypertension was primarily probably a neurological disease due to excessive neural discharge in efferent autonomic fibres to the blood vessels of the kidneys, and that a state of persistent vasoconstriction, when present in these vessels, resulted in the production of a vasopresser substance which was liberated into the blood stream. The vasoconstrictor supply to the kidneys comes from the tenth, eleventh and twelfth thoracic and first lumbar segments, and the kidneys may be denervated by section of the splanchnic nerves and the connexions of the sympathetic ganglia at these levels. Considerable improvement has been noted in cases in which the operation was restricted to one side. The symptoms for which operation was recommended were headache, dizziness, dyspnœa, insomnia, nocturia and failing vision, and it was suggested that the following criteria should be followed before operation was undertaken. The patient should not be over fifty years of age, and systolic blood pressure readings

as high as 200 millimetres of mercury should have been observed. The specific gravity of the urine should not be below 1010 and the blood urea nitrogen content should not be above a high normal level. Papilloedema, retinal hæmorrhages and exudates are not contraindications to operation, since excellent results have been obtained in cases in which these signs were present.

The present writer has recently operated on three patients with essential hypertension, and as the early results which have been obtained are considered to be reasonably satisfactory, these cases are reported in the present article.

Case I.

Mrs. R.L., aged forty-nine years, was referred by Dr. J. D. Herlihy on August 3, 1937. She was complaining of bilateral frontal headache which had commenced ten years before. At first the headaches would appear every three or four days, but in the previous twelve to eighteen months they had occurred every day. She was usually awakened between 3 a.m. and 5 a.m. by a headache which was fairly severe, dull in character and persisted until late in the morning. Recently she had experienced nausea and vomiting on lying down at night. Otherwise she was a healthy woman, had had no previous serious illnesses, performed her own household duties and did not become breathless on exertion.

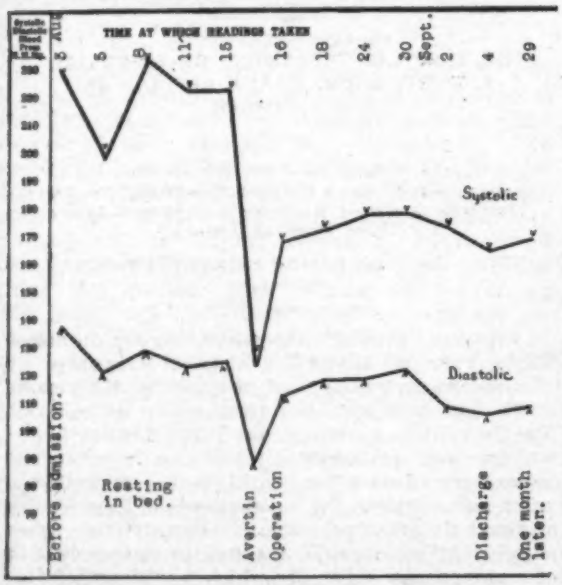

FIGURE I.
Graphic record of the blood pressure readings before and after operation in Case I.

The visual acuity was estimated at $^6/_6$ in each eye; the fundi were normal; the visual fields were full, and examination of the nervous system disclosed no abnormal physical signs. The apex beat was in the fifth intercostal space, four inches from the mid-line. The systolic blood pressure was 230 and diastolic 140 millimetres of mercury. Electrocardiographic examination showed "no significant changes in the record". X ray examination of the chest showed that "the cardiac shadow and the great blood vessels were not enlarged". The blood urea nitrogen content was 14·0 milligrammes per 100 cubic centimetres. The specific gravity of the urine was 1015 and the result of the urea concentration test was normal. There was a slight cloud of albumin occasionally present in the urine.

Before operation the patient was kept at rest in bed for fourteen days, during which period the general level of the blood pressure fell, and on one occasion readings of 220 (systolic) and 122 (diastolic) were made (see Figure I). At operation on August 16, 1937, the greater and lesser splanchnic nerves and the connexions of the sympathetic chain between the tenth and twelfth segments were sectioned on the left side only, after removal of portion of the eleventh rib. The operation was performed under "Avertin" anæsthesia and it was noted that the effect of the "Avertin" alone was to produce a fall in blood pressure to 125 (systolic) and 85 (diastolic). This fall was maintained for only a very short time, and within an hour and a half of the induction of the anæsthesia the pressure had risen to 195 (systolic) and 125 (diastolic). The effect of section of the autonomic nerves referred to produced a rise of diastolic pressure to 140 millimetres of mercury and a preliminary fall of systolic pressure, almost immediately followed by a secondary rise. The effect of the operation on the blood pressure can be seen in Figure I. The patient was discharged on September 4, 1937, with a systolic blood pressure reading of 166 and diastolic of 108 millimetres of mercury. In the three weeks succeeding operation she had had two mild headaches, one two days after operation and one four days after operation. This, she stated, was the longest period of freedom from headaches for several years.

When she was interviewed one month after discharge from hospital the systolic blood pressure was 170 and the diastolic 110 millimetres of mercury, and she stated that she had had no further headache.

Case II.

Mrs. J.F., aged fifty-five years, was referred to Lewisham Hospital by Dr. S. V. Marshall on July 8, 1937. This patient was a trained nurse and had worked hard all her life until a few months previously, when she had been compelled to relinquish her occupation as the result of her symptoms. She complained of the occurrence of attacks of precordial pain and pain in the legs during the previous six months, breathlessness on exertion during the previous four to six months, and occasional morning headache during the same period. She had lost 3·2 kilograms (seven pounds) in weight in the previous three months.

She was a healthy-looking, middle-aged woman with an extremely florid complexion. She was slightly breathless as the result of walking from the street to the hospital. The visual acuity of each eye was $^6/_6$, the fundi were normal and the visual fields full. Examination of the nervous system disclosed no abnormal physical signs. The apex beat was diffuse and could not be localized accurately. The systolic blood pressure was 224 and the diastolic 160 millimetres of mercury. The electrocardiographic report stated: "The change in the *T* wave in all leads is that associated with hypertension." The blood urea nitrogen content was 17 milligrammes per 100 cubic centimetres. Examination of the urine showed a specific gravity of 1015, a trace of albumin and an occasional epithelial cell. The result of the urea concentration test was normal.

The patient was rested for a fortnight in hospital before operation. During this time she had several attacks of precordial pain and pain in the lower limbs. The latter would sometimes wake her up at night and keep her awake. She stated that these attacks of pain were not necessarily associated with exertion and had often taken place when she was at rest. During this resting period in hospital the general level of the blood pressure fell (see Figure II), and on one occasion a pressure of 158 (systolic) and 100 (diastolic) was recorded; but on the day before operation the systolic pressure was 210 and the diastolic 120 millimetres of mercury. At operation on July 24, 1937, the splanchnic nerves and the connexions of the sympathetic chain between the tenth and twelfth dorsal segments were sectioned on the left side. Immediately on section of these nerves the same alterations of blood pressure were observed as described in Case I. On the day after operation the systolic blood pressure was 120 and the diastolic 80 millimetres of mercury. She had a severe cramp in the left leg twenty-four hours after

operation; but this was thought to be postural in origin. The changes in blood pressure following operation are seen in the records in Figure II. During convalescence some pleuritic pain occurred on the left side; this was relieved by the application of diathermy. A stitch abscess developed in the wound, and this delayed her discharge from hospital until September 13, 1937. At the time of her discharge the systolic blood pressure was 170 and the diastolic 120 millimetres of mercury. Her facial appearance has changed considerably following operation.

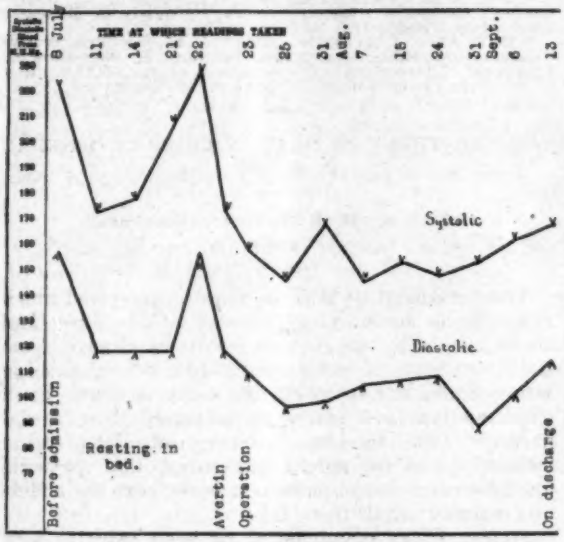

FIGURE II.

Graphic record of the blood pressure readings before and after operation in Case II.

She has lost the florid look and appears several years younger. In the two months following operation she has not experienced any of her previous symptoms and says that she feels healthier at the present time than she has felt for many years.

Case III.

Mr. J.W., aged thirty-eight years, a clerk, reported to Lewisham Hospital on June 30, 1937, and stated that he had had an attack of numbness and tingling on the left side, involving both the left arm and the left leg, six months previously. This attack had taken place on his rising from bed one morning. The numbness in the left hand and leg had persisted. Six weeks after this first attack he had an attack of paresis on the right side while going home from work one afternoon. Except for these attacks he had previously been a healthy man. He had had no serious illnesses, and led a very regular life; he did not drink or smoke and he had not suffered from headache, giddiness, insomnia, frequency of micturition or visual disturbance. One brother suffered from epilepsy. There was no family history of hypertension. He had not worked for one month past.

On examination the visual acuity of each eye was found to be 6/6. Examination of the fundi disclosed kinking of the veins at the points of arterial crossing; but the arteries were normal in calibre and were not tortuous. Papilloedema and hæmorrhages were not present in either fundus. Detailed examination of the nervous system disclosed no abnormal physical signs. The apex beat was in the fifth intercostal space, 9·4 centimetres (three and three-quarter inches) from the mid-line, and the first sound at the mitral area was prolonged and the second sound at the aortic area was accentuated. It was found that the systolic pressure could not be recorded, since the sphygmomanometer which was being used read only to 300 millimetres of mercury. On a second instrument the systolic pressure proved to be 315 and the diastolic 180 millimetres of mercury. At the date of examination he had no symptoms other than the persistent numbness in the left hand, very easily induced fatigue (a recent symptom) and slight breathlessness on exertion.

The blood count was normal, the blood urea nitrogen content was 20 milligrammes per 100 cubic centimetres, the specific gravity of the urine was 1016, and the result of the urea concentration test was normal. There was an occasional faint cloud of albumin in the urine.

The patient was admitted to hospital on July 12, 1937, and rested for one week before operation. During this week the general level of his blood pressure fell and the lowest reading was 284 (systolic) and 150 (diastolic). At operation on July 17, 1937, the splanchnic nerves and the connexions of the sympathetic chain from the tenth to the twelfth thoracic segments were sectioned on the left side. Under the "Avertin" anæsthesia the systolic blood pressure fell to 225 and the diastolic to 145 millimetres of mercury. After section of the nerves on the left side the reading was 210 (systolic) and 140 (diastolic).

During the week following operation the systolic pressure varied between 240 (maximum) and 190 (minimum). He was discharged from hospital on July 26, 1937. The pressure on discharge was 218 millimetres of mercury (systolic) and 136 (diastolic).

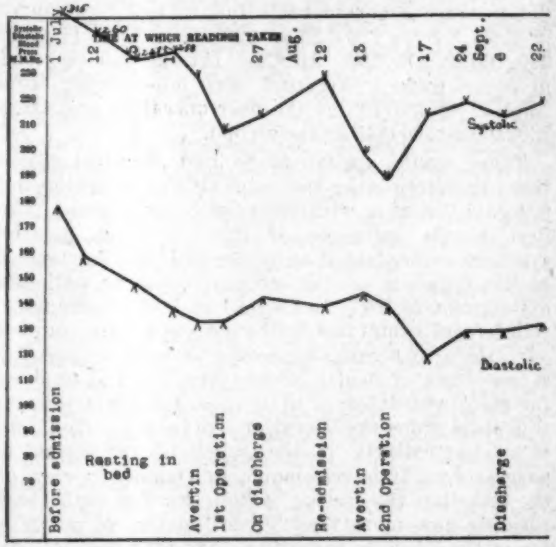

FIGURE III.

Graphic record of the blood pressure readings before and after operation in Case III.

He was readmitted to hospital on August 12, 1937, for operation on the right side. Operation was performed on August 13, 1937. In the week following the second operation the systolic pressure ranged from 175 to 220 millimetres of mercury. The patient reported back on September 19, 1937, and stated that he felt reasonably well and was getting about at home. The systolic blood pressure was 220 and the diastolic 136 millimetres of mercury. He had had a great deal of pleuritic pain following the second operation, and this had been relieved by diathermy. The paræsthesia in the left hand persisted.

Comment.

The criteria recommended by Peet[2] were followed in these three cases, excepting that in Case II the patient was a woman, aged fifty-five years. However, as she had led a regular, healthy

life, and since her age was the only departure in her case from the criteria recommended by Peet, it was decided not to refuse operation on this ground alone. In none of the cases was an attempt made to predict the results of the operation by observation of the fall of blood pressure following "Pentothal Sodium", as suggested by Allen,[3] since the occurrence of cardiac arrhythmia and extrasystoles have been reported following the use of this drug. The present writer feels that it would be unwise to submit a patient already suffering from cardiac overload to the possibility of these sequelæ, particularly as there is no certainty that the operation will produce a complete relief from symptoms even in the presence of a considerable fall of blood pressure following the use of "Pentothal Sodium". On the other hand, it has also been noted that in some cases a complete relief from symptoms follows operation without producing a fall in the blood pressure of more than 50 millimetres of mercury. It was noted in Cases I and II, reported here, that the induction of anæsthesia by "Avertin" did produce a considerable fall in blood pressure: in Case I from 220/122 to 125/85, in Case II from 230/160 to 175/120. In Case III the fall of blood pressure under "Avertin" was much less: from 250/140 to 230/135 at the first operation and from 280/160 to 200/145 at the second.

There would appear to be two chief considerations in determining the value of this operation on patients suffering with essential hypertension. The first is the influence of the procedure on the symptoms complained of by the patients. The second is the influence of the operation on the patient's expectation of life. In Cases I and II the dramatic relief from symptoms following operation, coupled with the gratification expressed by both of them for a new state of health which they had not enjoyed for many years, leaves little room for doubt that on this score alone the operation has been justified, and more particularly if the relief of symptoms is permanent. It is reasonable to assume, in view of the fact that the resting systolic level in both these cases is now more than 50 millimetres of mercury lower than before operation, that they are both in a better position as far as the likelihood of some vascular catastrophe is concerned than they were before operation. The patient in Case III was relatively symptomless; but as he had had two intracranial vascular disturbances before operation, it is reasonable to suppose that without treatment further similar attacks might be expected in the near future and that within the next twelve months a frank intracranial hæmorrhage would not be improbable. As his post-operative blood pressure (220/136) is considerably lower than that recorded before operation (315/180), it is not unlikely that a longer period will elapse under the present conditions before rupture of intracranial vessels takes place.

If one is justified in drawing any conclusion at this early stage from only three cases, it might be conservatively stated that, provided cases of essential hypertension are adequately selected for surgical treatment, the operation, which is a simple one and should not be attended by any mortality, is likely to produce some relief from symptoms and may lengthen the patient's life.

References.

(1) Gilbert Phillips: "Contemporary Neurosurgical Practice", THE MEDICAL JOURNAL OF AUSTRALIA, May 29, 1937, page 819.
(2) Max Minor Peet: "The Surgical Treatment of Hypertension", *University Hospital Bulletin*, University of Michigan, Ann Arbor, Volume III, January, 1937, page 1.
(3) E. V. Allen, J. S. Lundy and A. W. Adson: "Preoperative Prediction of Effects on Blood Pressure of Neurosurgical Treatment of Hypertension", *Proceedings of the Staff Meetings of the Mayo Clinic*, Volume XI, June 24, 1936, page 401.

THE TREATMENT OF HEAD INJURIES IN GENERAL PRACTICE.[1]

By R. A. MONEY,
Sydney.

THE treatment of head injuries has received much attention in recent years, almost *ad nauseam*; and there has been rather too drastic a change from the treatment of some years ago, when nothing was done, to one in which too much is done. Such emphasis has been placed on increased intracranial pressure, on cerebral œdema, on dehydration measures and on spinal punctures, that patients are now often found suffering more from the effects of treatment than from injury.

However, the management of head injuries may cause more anxiety than any other emergency in practice, and the prognosis in the early stages is very uncertain. Each case is a law unto itself, and each becomes an individual problem. None is too serious to be despaired of, and none too trivial to be neglected. The most important part of the treatment is that which concerns the injury to the brain and its commonest immediate effect, loss of consciousness, known as "concussion". Fracture of the skull is of little import, except that it implies a fairly severe injury, and may lead to hæmorrhage extensive enough to produce compression, or may open pathways for the entrance of infective organisms. Frequently the brain can be severely damaged and hæmorrhages can occur in the absence of any fracture.

The application of successful treatment will depend on a clear recognition of the various pathological states which may follow the injury, and which can be diagnosed only by careful and continued observation of the initial symptoms and signs and of any which arise later, especially during the "danger period" of the first forty-eight hours.

First-Aid Treatment.

The patient has usually been removed to the nearest shelter; and if he is in a state of severe collapse, further transportation should be delayed, and warmth *et cetera* should be applied. A dangerous degree of bleeding may be occurring from scalp

[1] Read at the annual meeting of the Northern District Medical Association (British Medical Association), held at Armidale on December 8, 1937.

wounds, which gape owing to retraction of the galea. This is best controlled with a bandage over a padded ring instead of a tourniquet. Suturing is not advisable until the wound has been cleansed and its depths have been explored, except as a last resort in severe arterial hæmorrhage. Search rapidly for associated fractures of the spine or limbs and for internal visceral damage, and give appropriate treatment. Do not give morphine, as it may mask important signs. Estimate roughly the degree of unconsciousness and note the relative size of the pupils and the paralysis, or otherwise, of one side of the face and the limbs. When removal to home or hospital has been decided upon, supervise the lifting personally, and see that all unnecessary rough handling and movement are avoided.

Examination and Observations in Hospital.

When the patient has reached hospital, the following procedure should be adopted.

The state of unconsciousness should be accurately estimated, classified in one of four grades, and subsequently recorded every hour according to the following headings: (i) drowsiness, which is indicated by response to a tactile stimulus, such as when the pulse rate and temperature are taken, or to a simple question such as "What is your name?"; (ii) stupor, which is indicated by response only to a shouted vocal stimulus or command, such as "Put out your tongue" or "Open your eyes"; (iii) deep stupor, which is indicated by response only to a painful stimulus; (iv) coma, which is indicated by failure to respond to all stimuli and absence of the corneal reflex.

Careful inspection should be made of scalp and cranium for signs of local trauma, and of the orifices and cavities of the orbits, nares, mouth and ears for blood and cerebro-spinal fluid.

The condition of the pupils and their reaction to light should be tested, and, if possible, the ocular fundi should be examined for swelling and hæmorrhages.

The existence of monoplegia, hemiplegia, spasticity, flaccidity and anæsthesia should be noted.

The state of the reflexes, abdominal, knee and plantar, and the presence or otherwise of ankle clonus should be recorded.

An hourly chart of the temperature (oral, axillary or rectal), the pulse rate, the respiration rate and, in certain cases, the blood pressure, must be kept. More will be said about the significance of these records later.

The action of the sphincters should be observed, especially the patient's ability to swallow and to micturate. Vomiting of blood may occur; and catheterization may be necessary for retention of urine.

X ray examination of the skull will rarely give any helpful information during the early stages, and should be postponed till the "danger period" is over. Good films can be obtained with the aid of a Lysholm grid when no Potter-Bucky diaphragm is available.

Early Treatment and Nursing.

The patient should be placed flat in a warm bed, in a quiet room, with the head turned to one side, and the airway unimpeded. It is generally better to use a small pillow than to extend the head fully. An intratracheal airway and oxygen or "Di-carbox" inhalations may be needed in bad cases. If mucus is troublesome the foot of the bed may be raised on blocks, and the discharge should be sucked out with a sucker and catheter as it drains into the pharynx and post-nasal space or cheeks. Four-hourly changes of posture from side to side or onto the back may be necessary to avoid pressure sores; but do not move the patient about roughly to undress. During the stage of cerebral shock no stimulants should be given, but reliance should be placed on rest, warmth, posture and fluids.

Scalp Wounds and Depressed Fractures.

Scalp wounds and depressed fractures need further attention as soon as the above measures and observations have been carried out and the patient's condition warrants it. Bleeding from scalp wounds may exsanguinate a patient quite rapidly. The hair should be cut or shaved away, and the floor of wounds should be inspected for fractures. Linear fractures may be neglected. Careful lavage with warm normal saline solution should then be performed to remove foreign bodies and blood clots, and *débridement* should be carried out. The lacerated edges should be excised cleanly and sutured. A soft rubber corrugated tube or "cigarette" drain may be left in for twenty-four hours.

Depressed fractures should always be dealt with where there is an overlying scalp wound, and a limited removal of the fragments should be carried out in order to avoid sepsis, by extirpation of the infected bone. If the overlying scalp is intact, operation should be undertaken only where stereoscopic X ray films reveal an indriven bone fragment or spicule, which may have penetrated the dura. In these cases there is a danger of further cerebral laceration or of hæmorrhage, if it has not already occurred. The other indication for immediate operation on a simple depressed fracture is when an associated extradural or subdural hæmorrhage is suspected owing to the situation of the fracture, or to the onset of signs of compression. Nothing should be done to the area from which bone is missing for a year, when it should be filled in with a bone graft from the inner table of the ilium, to prevent sequelæ and for psychological reasons.

Fractures of the Base and Prevention of Meningeal Infection.

Leakage of cerebro-spinal fluid, or blood, or both, from the nose or ears indicates a compound fracture of the base of the skull, with its attendant risk of meningitis. The nasal passages should be kept free from clots with hydrogen peroxide, and some antiseptic nasal drops should be instilled at frequent

intervals. The external ear should be swabbed, never syringed, with hydrogen peroxide and then an antiseptic, such as a 1 in 1,000 solution of perchloride of mercury in water; and then a small piece of gauze soaked in the same solution should be gently inserted and changed every hour.

In these cases there is always a real risk of meningitis supervening, even up to three or four weeks after the injury. Although the infecting organism is usually a pneumococcus, sometimes it is a hæmolytic streptococcus; accordingly, it is worth while giving prophylactic doses of sulphanilamide as soon as possible. A dose of two 0·5 gramme tablets *per os* may be given thrice a day if the patient can swallow, or ten cubic centimetres of "Prontosil Red" may be given intramuscularly daily if the patient cannot swallow. Should infection occur and a streptococcus be isolated, much larger doses will be necessary.

Posturing.

As previously mentioned, during the initial shock period the patient should be kept flat, with the head turned to one side. As recovery occurs, the head and shoulders should be elevated gradually, up to 30°. This is best accomplished in hospital on a special Lawson Tait type of bed; but the use of pillows and back-rests can achieve the same object. Care must be taken, however, not to elevate the patient before the systolic blood pressure has risen to 100 millimetres of mercury, or the intracranial pressure to over 110 millimetres of cerebrospinal fluid. After operations for the release of extradural or subdural collections of blood the intracranial pressure may remain low for a time, and, in order to aid the patient to recover consciousness, it may even be necessary to keep the foot of the bed elevated for some time. This was so in the following case.

A female, aged fifty years, slipped on a tiled veranda whilst hosing it and landed on her buttocks. She may have hit her occiput too. One week later she fainted whilst out walking, and was prostrated afterwards with persistent severe headache at the vertex, thought to be due to a menopausal syndrome. A week later she collapsed again, with extremely severe headache and hyperæsthesia of the scalp. This was followed by increasing drowsiness, thought to be due to the large doses of sedatives which had to be administered. On examination, great variability in the state of unconsciousness was noted, with bilateral third nerve palsy and early papilloedema. The pulse rate varied between 55 and 65 beats per minute. Both plantar reflexes were extensor. Lumbar puncture yielded faintly yellow fluid under a pressure of 260 millimetres of water, containing 80 milligrammes of protein per 100 cubic centimetres.

Under local anæsthesia a burr hole was made over each cerebral hemisphere, and a large left subdural hæmatoma was evacuated and drained, with immediate improvement. Three days later the patient had relapsed into deep stupor. The systolic blood pressure was found to be 105 millimetres of mercury, and the intracranial pressure only 80 millimetres of water. The foot of the bed was raised on nine-inch blocks, and consciousness was regained in thirty minutes. The patient's head was elevated two days later, but she became drowsy again. Thereafter she was kept lowered, and complete recovery took place in about ten days.

Fluids.

Fluids are needed by these patients, often badly, if much blood has been lost or if over-breathing is occurring. Very little is usually taken by mouth, and that only in spoonfuls, whilst from 1,500 to 2,000 cubic centimetres daily are necessary to prevent dehydration. The simplest and most efficient method is by intramuscular injection into the vasti muscles of the thighs; about 1,000 cubic centimetres of normal saline solution can be given daily by this route. Ringer's or Locke's solution may also be used. These solutions, and a complete apparatus for giving them, are available in convenient sterile containers holding one litre and called "Soluvac". Needless to say, the mouth and tongue need frequent cleansing with hydrogen peroxide as well as the usual glycerin of borax.

Control of Restlessness.

Restlessness and irritability may occur during the stage of cerebral irritation, and usually indicate laceration of the brain with diffuse subarachnoid hæmorrhage, the climax of which is seen in the so-called "fighting-drunk" stage, which may call for relief by spinal puncture. The bed should be screened off, or the room darkened. Rails should be put around the bed; but mechanical restraint should be avoided if possible. Good nursing may help tremendously. Make sure the bladder and rectum are empty. If all else fails, manacles and folded sheets may be necessary. Morphine depresses the hypothalamic and medullary nuclei after trauma, as well as masking a relapse into deeper unconsciousness. The ordinary mixture of aspirin, phenacetin and caffeine with potassium bromide should be tried first, or even a haustus of potassium bromide and chloral hydrate given by mouth or *per rectum*. The barbiturates are very valuable, especially as they are now available for intramuscular injection. A good dose of calomel, followed by magnesium sulphate to produce a liquid stool, will relieve congestion and headache tremendously.

Danger Signals.

During the expectant period of the first forty-eight hours and until consciousness is fully regained, the nurse should be instructed to call the doctor should any of the following "danger signals" be noted: (i) deepening of unconsciousness; (ii) slowing of the pulse rate to 60 or a rise above 140 beats per minute; (iii) a change to stertorous or irregular respiration; (iv) a rise of temperature above 39° C. (102·6° F.)—the point up to which "compensation" is taking place; (v) discrepancy in the size of the pupils; (vi) paralysis or paresis on one side of the body.

These signs indicate failing "compensation" and the onset of compression; and the doctor must decide quickly whether it is due to one of the varieties of progressive meningeal hæmorrhages, or to cerebral laceration or œdema, and whether operation is called for or not. Rapid progressive deterioration in the early stages after a severe injury does not necessarily call for surgery. These patients

belong to the group who are in deep stupor when first seen and either hemiplegic or quadriplegic. The corneal reflex is absent, and the pupils are dilated. The pulse is feeble, whether slow or rapid; and the temperature and respiration rate rise rapidly. Death occurs within the first forty-eight hours in hyperthermia. Nothing can save these patients, who succumb to extensive laceration of the brain and medullary compression.

The occurrence of a "lucid interval", or the maintenance of a stationary period of drowsiness or stupor, followed by a relapse to a deeper grade and accompanied by more than one of the above "danger signals", is strong presumptive evidence of meningeal hæmorrhage. If the interval has been of some hours' duration, the hæmorrhage is most likely to be extradural and unilateral from a middle meningeal artery; but if the interval has been of some days' or even some weeks' duration, the hæmorrhage is more likely to be subdural and often bilateral, because it arises from the cortical veins as they pass through the subdural space to join the longitudinal sinus. A waxing and waning of the state of consciousness and variable intense headaches are very characteristic of this latter condition, which is now recognized to be as common a cause of acute compression as the better known extradural hæmorrhage. Thus the exploratory operation, if decided upon, is not complete until the dura has been opened. However, remember that slowing of the pulse and even a unilateral dilated pupil with hemiplegia, in the absence of an increasing depth of unconsciousness, are not necessarily indications for operation, and may merely indicate cerebral damage, which will clear up if left alone.

Operation for Meningeal Hæmorrhage.

The technique of an emergency operation for meningeal hæmorrhage should be as simple as possible. No anæsthesia may be needed at all; or, at any rate, local infiltration with a 1% solution of "Novocain" should suffice. Through a vertical incision and a muscle-splitting approach in the temporal fossa, a burr hole is made in the temporal bone in the line of the anterior branch of the middle meningeal artery. Spinal puncture has usually been made beforehand to determine the exact intracranial tension and the presence or absence of blood in the cerebro-spinal fluid. A small mastoid retractor keeps the wound edges apart readily. Extradural hæmorrhage will be recognized at once. The bone opening should be enlarged and the clot should be sucked and washed out. If the actual bleeding point in the artery cannot be seen, occlude its main stem with a silk stitch, or plug the *foramen spinosum* with wax or a wooden match. If no extradural clot is found, a dark tinge will indicate a subdural hæmatoma, and a small incision in the dura will verify its presence. Subdural hæmorrhage remains fluid for a long time, on account of the osmotic passage of cerebro-spinal fluid through the endothelial covering, which rapidly surrounds it like a semi-permeable membrane. Thus it can be easily sucked and washed out through the small burr hole, and an osteoplastic flap need rarely be turned down. A second burr hole may be necessary in the parieto-occipital region; and through this the washing can be done. The opposite side should always be explored as well, as the condition is so frequently bilateral. A short soft rubber drain should be left in the epidural or subdural space for 48 hours or more, and the wound should be closed carefully.

The Control of Increased Intracranial Pressure by Spinal Puncture and Dehydration.

In cases in which operation is not indicated, and even after operation, the problem of control of intracranial pressure has to be considered. If at the end of forty-eight hours unconsciousness has persisted, or is perhaps deepening, if the blood pressure and temperature are rising and the pulse and respiration rates are altering, then the presence of progressive cerebral œdema or of subarachnoid hæmorrhage should be suspected, and spinal puncture should be performed. This is in order that the pressure of the cerebro-spinal fluid may be carefully and accurately measured with a manometer and the presence or absence of blood may be noted. If the initial pressure is above 120 millimetres of cerebro-spinal fluid, then fluid should be slowly run off until this level is reached. If the initial pressure is over 200 millimetres, and the fluid is clear, then active dehydration is indicated in addition, in order to prevent recurrence of the high pressure. If the patient can swallow, 7·8 grammes (two drachms) of magnesium sulphate in 60 cubic centimetres (two ounces) of water should be given by mouth, followed by 3·9 grammes (one drachm) in 30 cubic centimetres (one ounce) of water every hour till a watery motion is passed. Otherwise, the simplest plan is to run 180 cubic centimetres (six ounces) of 25% magnesium sulphate solution into the rectum, and to see that it is retained for at least half an hour. This procedure can be repeated in twelve hours.

If the initial pressure is over 300 millimetres and the fluid is clear, spinal puncture should be repeated daily in addition to the above procedures, until the pressure is within normal limits. Should the fluid be blood-stained, allow it to escape slowly till normal pressure is reached; but never apply any dehydration measures in the presence of active bleeding. This release of pressure and removal of blood-stained fluid give valuable relief in traumatic delirium after subarachnoid hæmorrhage. If the initial pressure is normal or low, then do not withdraw any fluid, do not dehydrate the patient, and do not place the patient in an elevated posture.

The rules for the use of intravenous injections of hypertonic solutions are more difficult to formulate. Their effect is transitory and is often followed by a greater reactionary rise of pressure. In high concentration, such as 100 cubic centimetres of 50% solution of glucose or sucrose, or 70 cubic centimetres of 15% to 20% sodium chloride solution,

they are very potent and must never be used if there is any evidence of active bleeding. In some cases, however, after operation, and when the pressure of the cerebro-spinal fluid is persistently above 200 millimetres despite two or three lumbar punctures, they are often beneficial; but then the use of 500 to 750 cubic centimetres of 10% glucose in normal saline solution is preferable to smaller and more concentrated amounts.

Finally, an occasional case of persistent localized cerebral contusion or generalized œdema will be found, in which the pressure and amount of cerebro-spinal fluid remain high, often over 300 millimetres, despite all these measures, and in the absence of a blood clot. Then simple subtemporal decompression will be needed, possibly bilateral, with a wide dural opening.

Hyperthermia.

The treatment of hyperthermia may be worrying during the initial stages and after operation. Up to 39° C. (102·6° F.) "compensation" is taking place, and there is no cause for alarm. Readings above that level are danger signals and indicate damage to the subthalamic or medullary centres. Apart from the appropriate necessary steps already described, the temperature must be reduced to avoid further damage to neurones. The following routine has been found effective. Starting with a sheet and two blankets, when the temperature reaches 37·7° C. (100° F.) cover the patient with only one blanket and one sheet; when the temperature reaches 38·8° C. (102° F.) cover the patient with only one sheet; when the temperature reaches 39·4° C. (103° F.) sponge the patient with tepid water; when the temperature reaches 40° C. (104° F.) sponge the patient with cold water; when the temperature reaches 40·5° C. (105° F.) sponge the patient with iced water; when the temperature reaches 41·1° C. (106° F.) cover the patient with cold wet sheets and expose him to a draught, or give continuous colonic irrigation with iced water till the temperature falls to 38·8° C. (102° F.).

Convalescence after Recovery of Consciousness.

The following remarks apply to the management of patients with minor head injury, who have had only a short period of consciousness, not observed by the doctor, or none at all, as well as to the type of case under discussion, after full consciousness has been regained. A new era in treatment now begins, in which the degree and duration of cerebral rest required can be gauged only by the presence and persistence of subjective symptoms. The chief symptoms are: (i) headache, (ii) giddiness on movement and stooping, (iii) inability to concentrate and work, and (iv) mental fatigue and insomnia.

So long as any of these persist the policy of strict rest and quiet should be enforced, assisted by the ordinary aspirin, phenacetin, caffeine and bromide mixture, with a barbiturate at nights as a hypnotic and a saline in the mornings as an aperient. The patient with severe headache should be allowed to assume the posture in which the greatest relief is obtained. Here, and in patients with other persistent symptoms, spinal puncture and manometry are invaluable and afford the only indications, whether to prop the patient up, to withdraw fluid and dehydrate the patient, or to keep him flat, stop dehydration and increase fluids. A "go-slow" yet optimistic policy must always be adopted, and lasting harm may be done by an attempt to shorten the convalescence and to allow the patient to return prematurely to work. It is far easier and wiser to alleviate all the symptoms by first intention, even if a little extra time is taken in treatment, because if they recur healing by second intention will be very much more difficult and will require a much longer relative time.

The following routine for patients cared for in their own homes or in private hospitals may afford some general indications, although, as I have previously stated, each case is a law unto itself not only according to the nature of the injury, but also to the nature and "make-up" of the injured head. The essentials are: (*a*) strict confinement to bed, (*b*) being read to rather than reading, (*c*) the exclusion of glare and noise, (*d*) the curtailment of visitors, and (*e*) the avoidance of boredom. If after this treatment the above-mentioned symptoms have been absent for three days or have not appeared three days after the recovery of consciousness, then the patient may be allowed some mental exercise and may sit up in a chair in the room for an hour or so. After a further two days' freedom from symptoms, the taking of a bath, visitors and unlimited reading may be allowed, and the midday meal may be taken sitting up in a chair. If after a final two days the patient is still symptom-free, a short walk or drive may be allowed, whilst attendance at meals and participation in the ordinary life of the household may be resumed. One, two or three weeks' holiday should be taken to allow a gradual progress to a full active life before work is resumed. The reappearance of symptoms at any stage must be the signal for a return to the point in the treatment at which the patient was symptom-free; and as a rule, the longer the initial period of unconsciousness, the more severe and prolonged will be the after-symptoms.

For patients treated in public hospital wards a somewhat similar but modified scheme can be followed; but the transition from the relative quietness of the hospital to the unsuitable conditions of the home is often too abrupt, and should be tempered by a period of two or three weeks in a well-conducted convalescent hospital.

In the type of case under discussion, if this plan is carried out it should be possible to avoid the bugbear of "traumatic neurasthenia" and the "post-traumatic syndrome", and to secure for the patient a return to work in about three or four weeks after the recovery of consciousness. Remember always that prevention is better and easier than cure. If no symptoms occur after consciousness is regained, proceed at once with the graduated resumption of

activities, lest too solicitous an attitude may sow the seed for a neurosis. On the other hand, give sympathetic attention to such symptoms as cause trouble, and recognize that an undue liability to fatigue, both bodily and mental, insomnia and lack of concentration are residual symptoms of organic damage and are not to be airily dismissed as neurotic.

The nature of the work to which the patient has to return must also be taken into account, and it is important that his full capacity should be tested before his recovery is declared complete. In some occupations, such as wharf-labouring and mining, there is no light work; and it is only asking for trouble to send such an injured worker back to light work when there is none to be done. In these cases a longer testing period off work till the patient is ready for full resumption will be required.

Patients who have never been unconscious after their injury, and who are symptom-free, should rest in bed for twenty-four hours and then return to their ordinary life. They should be warned, however, that delayed symptoms may occur and that they must report to their doctor should they do so. Treatment on the lines laid down should then be instituted. Indeed, a patient who has never had concussion may need as careful and prolonged treatment as one who has been unconscious for days. If, despite a rigid adherence to this routine during convalescence, signs and symptoms have persisted, and in mishandled patients if they have reappeared, further active investigation and treatment by spinal drainage, intravenous therapy, if the pressure is raised, and encephalography after air insufflation will be required; but this is a subject beyond the scope of this paper.

Bibliography.

C. P. Symonds and G. Jefferson: "The Treatment of Head Injuries", *The British Medical Journal*, October 12, 1935, page 677.
J. Fraser: "Head Injuries in General Practice", *The British Medical Journal*, April 10, 1937, page 637.
P. G. Flothow: "Observation on the Treatment of Head Injuries", *Northwest Medicine*, Volume XXXVI, Number 5, May, 1937, page 159.
R. A. Money: "The Management of Head Injuries", THE MEDICAL JOURNAL OF AUSTRALIA, December 14, 1935, page 810.

SOME ASPECTS OF THE LEPTOSPIROSIS PROBLEM IN AUSTRALIA.

By W. C. SAWERS,
Senior Medical Officer, School of Public Health and Tropical Medicine, University of Sydney.

THE description by Weil in 1886 of some cases of infectious jaundice is generally accepted as the earliest clear account of leptospiral infection, and the disease is still commonly known as Weil's disease. The causative organism was not discovered until 1914, when Inada and Ito recorded the discovery, in a case of Weil's disease occurring in Japan, of an organism now known as *Leptospira icterohæmorrhagiæ*. Subsequent to the elucidation of the cause, and hence the possibility of accurate diagnosis, increasing interest has been taken in this disease, and investigations have established its presence and importance in many countries of the world.

In the transmission and maintenance of the disease, two outstanding factors were early established. First, rodents, and in particular rats, serve as a reservoir for leptospiræ, as the organisms may persist in the kidneys of these animals during long periods, without causing their host any apparent material harm. Virulent leptospiræ, on excretion in the urine of these animals, can readily contaminate water and food. The second factor is the very frequent association of epidemics with water of a reaction suitable for the survival of these organisms.

Thus, from a public health aspect, the study of the problems, both difficult and interesting, raised by this chain is a matter of basic importance, if control of infection is to be attained.

Local conditions may produce special difficulties to be investigated; but of main import are the rat as reservoir and the infection of water. To mention shortly a few outstanding outbreaks of apparently diverse character but fundamentally with the same essentials, one may cite the outbreaks in the trenches during the Great War, the epidemics in the rice-fields of Japan, the infection of sewer workers, miners and fish workers in Great Britain, and the cases amongst bathers in Holland.

In Australia the history of leptospirosis is comparatively recent. In October, 1933, an outbreak occurred in the Herbert River district of North Queensland, of which Ingham is the principal town. These cases were diagnosed clinically by Dr. G. C. Morrissey and Dr. T. Leckie as Weil's Disease.[1] The laboratory aspects were investigated by officers of the Commonwealth Health Department and the causal organism was demonstrated in 1934, both from human cases and from rats.[2] [3] The outbreak was of interest, both from scientific and economic aspects, as it affected the cane industry of Queensland. Since then the laboratory aspects of the disease have received considerable attention, and of special interest are serological investigations by G. F. Lumley,[4] G. E. B. Clayton and E. H. Derrick,[5] D. W. Johnson, H. E. Brown and E. H. Derrick,[6] which have proved the presence of different serological strains in Queensland. Such serological difference of strains has also been noted abroad.

Among the very numerous problems of moment in leptospirosis, consideration of four only are attempted in this paper: (i) Nomenclature and classification; (ii) the rat reservoir, especially as regards Australia; (iii) the infection of water; (iv) the resistance of leptospiræ to adverse conditions.

Recorded in the consideration of these are the field observations and experiments carried out by an expedition, authorized by the Director-General of Health, Commonwealth Health Department, from the School of Public Health and Tropical Medicine, Sydney, to northern Queensland, in the latter part of 1934. In addition, certain laboratory experiments

have been carried out at the School of Public Health and Tropical Medicine, Sydney.

NOMENCLATURE AND CLASSIFICATION.

Consideration of nomenclature and classification raises definite problems with the vexed question of disease nomenclature, and these are enhanced when the leptospiral group, which is extensive and incompletely studied, is being dealt with. The term Weil's disease has received the sanction of custom, but is not satisfactory, as the causal organism was not known at the time of Weil's description. Other synonyms are acute infective jaundice, spirochætal jaundice, spirochætosis, seven-day fever of Japan *et cetera*. The term jaundice is undoubtedly misleading, as jaundice is not always, nor indeed generally, present in leptospirosis. Leptospirosis appears to be the most satisfactory name for this disease or group of diseases.

At present more precise classification would appear to rest on serological differences in the various strains of this group. Undoubtedly, this serological work, though painstaking and difficult, is of importance for classification purposes, diagnosis, and, very possibly, for serological therapy.

Although there is definite serological distinction between leptospiral strains, it remains to be seen whether serological criteria are sufficiently stable and reliable to render practicable the multiplication of named strains on such grounds. Hindle[7] notes:

> . . . that Fletcher isolated six serologically distinct groups of spirochætes from patients in Malaya, all showing very similar clinical symptoms, therefore there seems to be little, if any, difference between *S. pyrogenes* and other strains of pathogenic leptospira as regards the effects produced in human beings.

The general name of leptospirosis, supplemented when possible with the presence or absence of serological evidence of strain, appears the best solution in our present state of knowledge.

THE RAT RESERVOIR.

Animals vary in their susceptibility to infection by pathogenic leptospiræ. Guinea-pigs are extremely susceptible, and, in general, become jaundiced and die, showing typical *post mortem* appearances, the hæmorrhagic lesions being particularly well marked. The Pomona strain investigated by Derrick[8] is exceptional in exhibiting low virulence to guinea-pigs, twenty of twenty-two animals surviving infection.

Man, when infected, suffers a disease of varying severity, according to the virulence of the organism, the mortality in infection with some strains being at least as high as 30%, but in the Ingham outbreak probably below 5%. In convalescent human patients the organism may survive in the kidney and be found in the urine for weeks or months.

Some animals, as the rat, are apparently unaffected by the leptospiræ, and can act as reservoirs or carriers of these organisms, which persist in the kidneys and are passed out with urine. In this way the infection of water, food *et cetera* is effected by these animals.

Table I shows the results of the examination of a number of rats in north Queensland during 1935. The examinations were made by microscopic examination of kidney tissue under dark-ground illumination. The table includes the results of a number of examinations by the staff of the Commonwealth Health Laboratories additional to those made by the members of the investigating party.

A consideration of Table I, even when it is borne in mind that the numbers are in some instances very small, brings certain points of interest to notice.

Outstanding is the very striking association of *Rattus culmorum* with the outbreak of leptospirosis in the Herbert River district; in view of its importance, this rat will be considered in detail later.

In a brief review of the other species of rats, twenty specimens of *Rattus norvegicus* were examined; eighteen of these came from Townsville and none was obtained from the Ingham area. No leptospiral infection was found in this small number; but overseas experience has shown that, in general, *Rattus norvegicus* is the common carrier of leptospiræ. From personal experience and inquiry it seems that this rat does not frequent the cane-fields in the Ingham area, but is found in the larger seaports.

TABLE I.

Results of Examination of Rats for Leptospirosis.

Species of Rat.	Ingham.		Townsville.		Ayr.		Tully.		Innisfail.		Cairns.		Total.	
	Number Examined, 22/5/35 to 1/11/35.	Infected with Leptospiræ.	Number Examined, 7/1/35 to 1/11/35.	Infected with Leptospiræ.	Number Examined, 7/1/35 to 1/11/35.	Infected with Leptospiræ.	Number Examined, 7/1/35 to 1/11/35.	Infected with Leptospiræ.	Number Examined, 7/1/35 to 1/11/35.	Infected with Leptospiræ.	Number Examined, 26/9/35 to 20/10/35.	Infected with Leptospiræ.	Number Examined.	Infected with Leptospiræ.
Rattus rattus	31	0	16	1	2	0	11	0	1	0	40	0	101	1
Rattus culmorum ..	178	15	0	0	0	0	0	0	0	0	0	0	178	15
Rattus norvegicus ..	0	0	18	0	1	0	0	0	0	0	1	0	20	0
Melomys littoralis ..	1	0	0	0	1	0	0	0	3	0	0	0	5	0
Hydromys chrysogaster rapinus (water rat)..	1	0	0	0	1	0	0	0	0	0	7	0	9	0
Uromys caudimaculatus (water rat) ..	0	0	0	0	0	0	0	0	0	0	1	0	1	0
	211	15	34	1	5	0	11	0	4	0	49	0	314	16

NOTE.—One infected rat obtained from Innisfail previous to 7/1/35 and one from Cairns previous to 26/9/35.

Specimens of *Rattus rattus* examined numbered 101, and of these thirty-one were obtained from Ingham and the surrounding district. It is to be noted that, though not so heavily infected as *Rattus culmorum*, in view of its ubiquitous habits, this rat may possibly prove of distinct importance in the spread of leptospirosis from local centres of heavy infection.

The following quotation from Topley and Wilson[9] gives an idea of rat infection overseas:

Infection of Rats.—It has been established by numerous workers that the rat—particularly *Rattus norvegicus*—is a very frequent carrier of pathogenic leptospiræ. In Japan 40·2 per cent of 149 rats of this species were found infected (Ido et al. 1917a); in England 30 per cent of 100 wild rats (Balfour 1922, Stevenson 1922); in Scotland 36·7 per cent of 166 wild rats (Buchanan 1927); in Berlin 10 per cent. of rats (Uhlenhuth and Zuelzer 1921); in Toronto 37 per cent (Cameron and Irwin 1929); in Freiburg 44 per cent (Zimmermann 1930); in Moscow 12 per cent (Ssinjuschina 1929); and in Kiew 31 per cent (Basilewsky 1933). In Rotterdam, where cases of Weil's disease are fairly common, from 7 to 40 per cent of all rats examined have proved to be infected (Schuffner 1934). The frequency of infection depends largely on the age of the rat. Young rats are seldom infected, while over 50 per cent of adult rats may harbour the spirochætes. The black rat rarely acts as a carrier. Field-mice are not uncommonly infected (Ido et al. 1917a, Buchanan 1927).

In Australia, apart from northern Queensland, the question of rat infection has been considered by Johnson, Brown and Derrick,[10] who state:

In a series of eighty-five wild rats examined in Brisbane in 1936, two were found to be carriers of leptospiræ. In a second series of one hundred rats examined in March, 1937, six were found to be carriers of leptospiræ. The two strains of leptospiræ isolated from rats in 1936 were found to correspond serologically with the Wijnberg strain of *Leptospira icterohæmorrhagiæ*. The human strain isolated, while in general similar to the others, showed some relationship also to an Andaman strain. This is the first time that cases of classical Weil's disease and the specific type of leptospira have been recorded in Australia. The presence of *Leptospira icterohæmorrhagiæ* in Brisbane sewer rats, and the accompanying liability of sewer workers to become infected, brings Brisbane into line in this respect with many other cities of the world. The carrier rate is, however, comparatively low, and only sporadic cases of Weil's disease are likely to occur.

Other interesting considerations and findings are also discussed in the article by Johnson, Brown and Derrick.

In Sydney a number of live rats were obtained between February 21, 1935, and May 13, 1937, and have been examined at the School of Public Health and Tropical Medicine with the results shown in Table II.

It will be noted that microscopic examination of the kidneys of 102 rats examined under dark-ground illumination revealed no leptospiræ, and these findings in 37 cases were confirmed by guinea-pig inoculation. It is thus unlikely that at present there is any widespread infection of Sydney rats.

Of the other rarer species examined in north Queensland, the indigenous rat *Melomys littoralis* is found in the cane-fields of the Ingham district, and is of economic importance as a damager of cane. Being a climbing rat it eats the cane well above the ground and causes the breaking down of a larger proportion of stalks per individual than the culmorum rat. The number of these rats captured and examined is as yet small, and no significant evidence is as yet available of material leptospiral infection. This species is described by E. le G. Troughton (in *The Records of the Australian Museum*, Volume XIX, Number 4, September, 1937), and to Mr. Troughton we are indebted for the identification of those rats whose species was in doubt. A few large water rats and one large white-tailed rat were captured and examined, but were not found to be infected.

FIGURE I.
Rattus culmorum.

Rattus Culmorum.

As the culmorum rat is the main reservoir of infection in cane-field infections, full information of its habits is of importance for preventive measures.

TABLE II.
Results of Examination of Rats Captured in Sydney between February 21, 1935, and May 13, 1937.

Source.	Number.	Results.	Classification.		
Board of Health	16	No leptospiræ found by dark-ground examination. Material from 37 injected into guinea-pigs produced no infection.	*Rattus rattus.*	*Rattus norvegicus.*	Total.
City Council	75		35	27	62
Quarantine Station	2				
School of Public Health and Tropical Medicine	7		Young.	Adult.	Total.
Sydney home	2				
Total	102		47	34	81

The original description of *Rattus culmorum*, by Thomas and Dollman, is in *The Proceedings of the Zoological Society of London* (1908). An account of its habits was not available until the paper by K. R. Gard, "The Rat Pest in Cane Areas", was contributed to the fifth congress of the International Society of Sugar Technologists, Brisbane, 1935. Owing to Mr. Gard's extensive experience of the Ingham area, his paper is of particular interest. Space permits of only a few quotations of interest, as follows:

This species is usually larger and clumsier than either the Tree or House Rat, its general build suggesting ground movement rather than climbing, although it undoubtedly does climb quite well. The tail is short, bristly and non-prehensile, while the feet pads are less developed than those of the other two species. It is essentially a burrowing animal and mainly nests underground. It is a good swimmer.

General Habits.—Its habitat is the field, though it is not peculiar to cane fields and their environs, as it has been found up to 50 miles distant therefrom. It is nocturnal and avoids clear open spaces, through fear of natural enemies; it favours thick grassy harbouring places. The presence of water near the burrows does not appear important, as large numbers are frequently found even in dry periods away from water.

Food Habits.—Although the field rat may also be termed omnivorous, its main items of diet are derived from plants. Seeds of all descriptions are most favoured, followed closely by the stem tissues, mainly of grasses. Sugar cane is only incidental and not all-important in its diet. It is also carnivorous, often eating the carcasses of its trapped fellows. There has also been some evidence of its killing and devouring the tree rat. No attempt will be made to give a complete list of all the foods we have found to be eaten, but amongst the more prominent (besides sugar cane) might be mentioned the seeds of Sensitive Plant (*Mimosa pudica*), Noogoora Burr (*Xanthium strumarium*), Cowpears (*Vigna* sp.), Guinea Grass (*Panicum maximum*), Para Grass (*Paricum muticum*), wild Passion Fruit (*Passiflora herbertiana*), and the stem tissues of Para Grass, Blady Grass (*Imperata arundinacea*) and Cowpeas. The interior is the portion of the seeds devoured, the outer skin being generally discarded.

Breeding.—During the latter portion of 1934 and the early months of the present year we had the opportunity to study these rats during a particularly hot dry period of three months, followed by over a week's heavy rainfall and a subsequent period of showery conditions. Towards the end of the dry period we found that the rats were definitely not breeding; during this period over 200 females were examined and none were found to be pregnant. Soon after the rain began most of the females examined were pregnant. Of 273 examined throughout the wet period, 263 or 96·3 per cent were carrying young, whilst latterly a number of nursing females were again pregnant, this being observed in 39 instances. The maximum first litter found in these examinations contained 11 and the minimum 4, with an average of 7·5. The second litters were smaller, rarely exceeding 6.

It appeared that many females became pregnant at an age of less than two months. When in captivity females giving birth to young have almost invariably eaten them, but we have no data to show whether this happens to any extent in the natural state. We have the impression that this rat continues to breed throughout the year so long as weather conditions are favourable; but such breeding is arrested by dry conditions and the shortage of suitable young green food.

As seen in Table I, in this series of rats *Rattus culmorum* was obtained only from the Herbert River area surrounding Ingham. This is most probably due to the fact that the rats sent from other places were mainly town and not field rats. W. A. McDougall, in a paper "Identification of Rats Damaging Cane in Queensland Canefields" (*Cane Growers' Quarterly Bulletin*, April 1, 1936), states that specimens of *Rattus culmorum* have been identified from Ingham, Ayr, the Innisfail district, and from the Habana area of Mackay (November, 1935); and that the known localities infested with *Melomys littoralis* are Cairns, Ingham and Ayr. In respect to *Rattus norvegicus*, McDougall makes the following interesting observation:

Rattus norvegicus, the species which is considered as being of considerable importance as a pest of cane in Hawaii, has not been reported, as yet, as damaging cane in Queensland, although it is present in cities of the State.

FIGURE II.
Cane that has been gnawed by rats.

Breeding Experiments.

As many unsolved problems were apparent in respect of these rats and their infection with leptospiræ, an attempt was made to obtain a number for breeding purposes. Young rats were selected, as it had been noted by workers in Queensland, as also overseas, that leptospiræ are far more frequently found on microscopic examination by dark-ground illumination in mature than in young rats; and an uninfected strain was desired.

Of 40 young rats from the Herbert River district, 20 were selected for breeding and the remainder were killed, examined, and found by microscopic

examination to be uninfected. These rats were collected late in October and early in November, 1935. Of 20 rats brought to Sydney in December, 1935, seven (four males and three females) were living on January 17, 1936, when breeding records were commenced, the remainder having died in transit or in Sydney prior to this date. Eleven of these were available for examination, and six were found to be infected with leptospiræ (verified in four cases by guinea-pig inoculation). The history of the seven breeding rats is as follows:

One died aged approximately nine months; leptospiræ were present in the kidneys.
One died aged approximately eleven months; no leptospiræ in kidneys.
One died aged approximately fourteen months; no leptospiræ in kidneys.
Two died aged approximately eighteen months; no leptospiræ in kidneys.
Two killed aged approximately twenty-two months; no leptospiræ in kidneys.

Thus of 18 rats examined of the original stock of 20, in seven leptospiræ were found in the kidneys (approximately 39%). The breeding results are shown in Table III. Many of the litters disappeared

TABLE III.
Results of Breeding Experiments.

Number of Progeny in Litter.	Number of Litters.	Total Number of Progeny.
2	3	6
3	7	21
4	13	52
5	7	35
6	9	54
7	4	28
8	4	32
9	1	9
11	1	11
14	1	14
Total	50	262

Average size of litter: 5·2.

shortly after birth and had apparently been eaten. Eighty-one of the progeny were found dead, aged between 5 and 28 weeks, and of these, 43 were examined for leptospiræ and found to be uninfected.

TABLE IV.
Particulars in Regard to Size and Weight of Rats at Different Ages.

Age in Months.	Size.			Weight.		
	Length of Body in Centimetres.	Length of Tail in Centimetres.	Ratio of Tail to Body.	Male in Grammes.	Female in Grammes.	Average Weight (Male and Female).
3	11·0	7·0	0·64	70	45	58
6	13·5	8·5	0·63	88	63	76
15	—	—	—	151	107	129

A mixture of the following is kept in the pens:

Pollard	8 pounds
Maize meal	6 pounds
Oatmeal	4 pounds
Meat meal	1 pound
"Vita B"	½ pound

In addition, carrot, dandelion and whole-meal bread are given occasionally.

The average size of litter was smaller than that found by Gard under natural conditions; but the result, that from an original stock of three females in some eighteen months 262 young resulted, illustrates the increase that can be expected under favourable conditions.

Of major importance is that, of the 40 young rats originally obtained, 20 killed when young proved by microscopic examination to be uninfected, while 7 out of 18 of the remaining 20, preserved for breeding, later proved to be infected. This may well indicate that infection takes place early in the rat, but is not evident by microscopic examination of the kidney until later. It must be noted, however, that the growth of these animals is rapid. Donaldson[11] states that in the white laboratory rat sexual maturity, as indicated by the structure of the gonads, usually occurs in both males and females at the age of about two months or less, and Gard considers that many female culmorum rats became pregnant at an age of less than two months.

Mode of Infection of Rats.

In an attempt to elucidate possible ways of rat infection, an experiment involving 150 white rats was planned. Infection by drinking, feeding and percutaneous, conjunctival, subcutaneous and intraperitoneal inoculation, with adequate controls, was attempted. The rats were killed at weekly intervals and carefully examined.

Some of the rats inoculated intraperitoneally, subcutaneously and through the abraded skin became infected, but those inoculated by other methods did not.

The experiment was inconclusive, as the white laboratory rat is relatively refractory to leptospiral infection; but it was shown, as recorded elsewhere, that infection could be achieved, and, indeed, a case has been reported of the infection of a laboratory assistant following the bite from a white rat.

Infection in the Culmorum Rat.

Since *Rattus culmorum* is of particular interest, in view of its heavy infection in the Herbert River district, and is a local Australian rodent, experiments were projected, as supplies permitted, to ascertain possible modes of infection. The results are shown in Table V. These preliminary experiments showed that it was not as easy as expected to obtain a large percentage of infections by laboratory methods. The earliest positive kidney infection was detected by animal inoculation on the fourteenth day. Fourteen control animals were not infected, confirming the freedom of the laboratory-bred animals from leptospiral infection.

It is not yet clear how rat infection takes place, and further experiments are in progress. Intrauterine infection may be possible; but a greater percentage of infected rats might be expected if this were so. Also to be tried are further tests with infected water and the possibility of flea transmission during the period when leptospiræ may be present in the blood stream.

TABLE V.

Results of the Inoculation of Rattus Culmorum by Various Methods.

Route of Inoculation.	Number Inoculated.	Number Infected.	Number Uninfected.	Age of Rat in Months.	Remarks.
Subcutaneous	13	4	9	3 to 6	Examinations were made at weekly intervals from the first to the sixth week. The first positive result was detected by animal inoculation on the fourteenth day, the second and third microscopically on the fifteenth and sixteenth days; the fourth was detected by animal inoculation on the sixteenth day, though microscopic examination revealed no leptospiræ.
Intraperitoneal	10	2	8	3 to 10	Examinations were made at weekly intervals from the first to the fifth week. The first positive was detected by animal inoculation on the twenty-first day, the second by microscopic examination on the twenty-fourth day.
Abraded skin	10	2	8	5 to 6½	Examinations were made at weekly intervals from the second to the fifth week. Two positives were detected by animal inoculation on the third and fifth weeks. One rat examined after seven weeks and two after seventeen weeks were found to be uninfected.
Eye	3	0	3	5	As the above experiments showed few positive results before the third week, examination of the rats was commenced after the third week and continued at weekly intervals. None proved to be infected.
Total	36	8	28		
Controls	14	0	14		Eight of these were examined by guinea-pig inoculation as well as microscopically.

The Agglutination Test as a Means of Diagnosing Leptospiral Infection in Rats.

A short trial of agglutination tests as a means of diagnosing leptospiral infection was made on culmorum rats from an infected area, and indicated that a slightly higher percentage of positive results was obtained by this method than by microscopic examinations. This method is, however, more difficult, and is complicated by the possibility of the presence of more than one serological strain.

Taylor and Goyle[12] record somewhat similar results: "It has been noted in one area that while the carrier rate in rats was 40%, serological evidence of leptospiral infection was found in 60%." Thus the actual percentage of rats that have been infected may be considerably higher than is shown by microscopic examination; but this does not necessarily mean that all rats whose serum agglutinates leptospiræ are actually excreting leptospiræ.

The Infection of Water.

As the presence of water as well as that of rats has been associated with leptospirosis in most outbreaks in all parts of the world, field investigations of this aspect of the problem were carried out in the Ingham area. Moisture is essential for the survival of leptospiræ, and these organisms are very sensitive to the hydrogen ion concentration of the medium in which they grow. Hindle[13] notes:

> On the east coast of Sumatra, leptospira are found in every kind of water and cases of Weil's disease occur the whole year round. In the neighbouring island of Java the waters on the west coast were found to be almost free from leptospira and Weil's disease is very rare. This presence or absence of leptospira was correlated with differences in the pH of the water. The optimum seems to be about 7·3 to 8·0 at a temperature of 28° to 30° C., conditions realized in Sumatra but not in Java, where the pH of most of the waters was about 6·0.

Our investigations were carried out in October, 1935, during an extremely dry period, and with the exception of watercourses and ponds, all surface

FIGURE III.

Stagnant stream, showing density of the surrounding vegetation. (Location of Sample 1.)

water had dried off and a considerable area of country had to be covered to obtain data. The method employed was the testing of representative samples of water by the Hellige comparator, with

permanent coloured glass standards, and readings were made immediately after the taking of the sample. The pH readings varied from below 6·0 to 7·7.

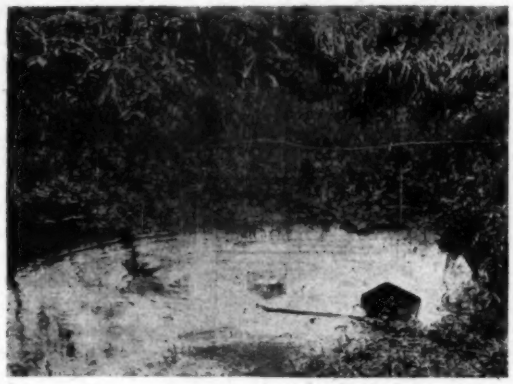

FIGURE IV.
Guinea-pigs exposed in water at Location 2.

Animal Experiments.

Additional to the testing of the reaction of samples of water, attempts were made to obtain guinea-pig infection by the immersion of scarified animals for up to 45 minutes in the suspected waters, and also by intraperitoneal inoculation into guinea-pigs previously inoculated with gas gangrene antiserum. Of nineteen animals, two only became infected, and these were scarified animals that were immersed in a stagnant stream adjoining cane-fields that would drain into the stream. It is very significant that the pH of this water was 7·7, and therefore suitable for the survival of leptospiræ. The infecting organism was obtained in pure culture, and subcultures proved virulent to guinea-pigs.

FIGURE V.
Fully grown cane, showing potential rat harbourage.

In comment, the use of a portable comparator would appear to offer a quick and simple method of locating water supplies that are suitable for leptospiral growth and are thus potentially dangerous in areas where infected rats are prevalent. The question is also raised of the possibility of altering the reaction of the water to an extent to make it unsuitable for the survival of leptospiræ, by the direct addition of chemicals or by the choice of a suitable fertilizer.

The problem is difficult and involved and is considered by Taylor and Goyle,[14] as regards both calcium cyanamide as a fertilizer and the addition of sulphuric acid to water in suitable proportions.

THE RESISTANCE OF LEPTOSPIRÆ TO HEAT.

Laboratory Test.

A fourteen-day culture of leptospiræ, originally isolated from a human case at Ingham, was used.

TABLE VI.

Effects of Heat on Leptospiræ.

Experiment.	Time of Leptospiræ in Bath.	Temperature of Bath.	Result of Dark-ground Microscopic Examination.
1	10 minutes.	50° C.	Leptospiræ inactive.
2	10 minutes.	45° C.	Leptospiræ active.
3	1 minute.	50° C.	A few leptospiræ feebly active.
4	1 minute 20 seconds.	50° C.	Leptospiræ inactive.
5	Control tube.	Unheated.	Leptospiræ active.

Sterile, sealed capillary tubes containing this culture were prepared and immersed in a water bath. Immediately after removal from the bath, the tubes were cooled in tap water at room temperature. The results obtained are shown in Table VI.

Field Experiments.

Arrangements were made to attend the burning of a cane area. The weather had been showery and the "burn" obtained was moderate. Four metal thimbles were wired to a suitable cane stalk at heights 175 centimetres (five feet ten inches), 105 centimetres (three feet six inches), and 15 centimetres (six inches) from the ground. These thimbles contained the following:

(*a*) Benzoic acid (melting point, 120° C.).
(*b*) Salicylic acid (melting point, 158° C.).
(*c*) Amygdalin (melting point, 215° C.).
(*d*) Tin foil (melting point, 232° C.).

A maximum and minimum thermometer was buried to a depth of 2·5 centimetres (one inch) at the foot of the cane stalks. Two small Gooch crucibles, containing leptospiral cultures, were placed in a dish filled with earth, and these were buried to surface level.

After the burning, the heat of which was sufficient to compel observers to stand ten to fifteen yards away, it was found that: (i) the contents of all the thimbles had volatilized or had melted, (ii) the reading of the buried maximum and minimum thermometer was 31·7° C. (89° F.), (iii) the crucibles contained burnt débris and a small amount of culture fluid. The latter was later inoculated into guinea-pigs with negative results; but as the amount (0·5 cubic centimetre) was small the result cannot be regarded as conclusive.

FIGURE VI.
Burning cane, showing clearing effected by burning.

At a second burning the maximum and minimum thermometer was placed on the ground but not buried. The result showed a reading of 76·7° C. (170° F.), the limit of the thermometer.

In comment upon these results, and from actual observation of cane burning, it appears that the heat generated is considerable, but of so short duration that, while almost certainly sufficient to destroy leptospiræ on the cane itself and probably on the surface layer of the soil, it would not appreciably penetrate either soil or water.

SUMMARY.

1. Difficulties in nomenclature and classification are briefly considered.

2. The results of field observations in north Queensland are recorded, with special reference to problems of rat and water infection and resistance of leptospiræ to heat.

3. An account is given of a successful attempt to establish the culmorum rat under laboratory conditions, and the results of some preliminary experiments in rat infection.

4. Attention may well be drawn to the increasing recognition of leptospiral infection throughout the world, and the probability that in Australia this disease is not limited to Queensland, and that infection of rat and man will be found in other States of Australia.

ACKNOWLEDGEMENTS.

The results recorded represent the work of the members of the investigation party, which consisted of Dr. A. H. Baldwin, Mr. F. H. Taylor, Mr. F. R. Barrett and the writer; and at the School of Public Health and Tropical Medicine, Sydney, Mr. A. R. Tremain, B.Sc., and Mr. J. Lawrence in particular were responsible for much valuable and painstaking work.

To Professor Harvey Sutton we are indebted for facilities and help, both in field and in laboratory work.

Dr. Paul Mitchell, Chief Quarantine Officer (General), North-Eastern Division, and medical officers and personnel of the Commonwealth Health Laboratories at Cairns and Townsville extended us every facility and aid. Sir Raphael Cilento, of the Queensland Health Department, very kindly afforded us the cooperation of his department. During our work at Ingham we had, as previously, the generous cooperation of Dr. C. G. Morrissey, Dr. T. Leckie, Mr. Myles Kelly and Mr. J. Fisher.

We have also to acknowledge in general terms assistance from many other sources, and it is regretted it is not possible to make individual acknowledgement.

REFERENCES.

(1) C. G. Morrissey: "Report on the Occurrence of Leptospirosis (Weil's Disease) in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, October 13, 1934, page 496.

(2) T. J. Cotter and W. C. Sawers: "A Laboratory and Epidemiological Investigation of an Outbreak of Weil's Disease in Northern Queensland", THE MEDICAL JOURNAL OF AUSTRALIA, November 10, 1934, page 597.

(3) T. J. Cotter: "Weil's Disease in North Queensland", paper presented at the one hundred and third annual meeting of the British Medical Association, Melbourne, 1935.

(4) G. F. Lumley: "Leptospirosis in Queensland: A Serological Investigation Leading to the Discovery of Distinct Serological Groups of Leptospiræ Causing Leptospirosis as it Occurs in Northern Queensland, with some Other Related Observations", THE MEDICAL JOURNAL OF AUSTRALIA, May 1, 1937, page 654.

(5) G. E. B. Clayton and E. H. Derrick: "The Presence of Leptospirosis of a Mild Type (Seven-Day Fever) in Queensland", THE MEDICAL JOURNAL OF AUSTRALIA, May 1, 1937, page 647.

(66) D. W. Johnson, H. E. Brown, and E. H. Derrick: "Weil's Disease in Brisbane", THE MEDICAL JOURNAL OF AUSTRALIA, May 29, 1937, page 811.
(67) E. Hindle: Chapter entitled "The Leptospiroses", the Medical Research Council's "System of Bacteriology", Volume VIII, page 314.
(68) G. E. B. Clayton and E. H. Derrick: "The Presence of Leptospirosis of a Mild Type (Seven-Day Fever) in Queensland", THE MEDICAL JOURNAL OF AUSTRALIA, May 1, 1937, page 647.
(69) W. W. C. Topley and G. S. Wilson: "The Principles of Bacteriology and Immunity", Second Edition, 1936, page 1447.
(70) D. W. Johnson, H. E. Brown, and E. H. Derrick: "Weil's Disease in Brisbane", THE MEDICAL JOURNAL OF AUSTRALIA, May 29, 1937, page 811.
(71) H. H. Donaldson: "Memoirs of the Wistar Institute of Anatomy and Biology, No. 6, The Rat", page 18.
(72) J. Taylor and A. N. Goyle: "Leptospirosis in Andamans, with Appendix on Present Knowledge of Leptospiral Infections", *Indian Medical Research Memoirs*, Number 20, March, 1931.
(73) E. Hindle: *Loco citato*, page 300.
(74) J. Taylor and A. N. Goyle: *Loco citato*.

Reports of Cases.

REGIONAL ENTERITIS.

By DAVID B. SKEWES, M.B., B.S. (Melbourne),
Surgical Registrar, Perth Hospital.

FOLLOWING an article on regional enteritis by Arnold S. Jackson in *Surgery, Gynecology and Obstetrics*, July, 1937, the report of a case, presenting appearances almost identical with the coloured photograph of this condition in that issue, may be of interest.

As no reference to this rare condition has appeared in previous issues of THE MEDICAL JOURNAL OF AUSTRALIA, it is probable that many Australians are not aware of it, and a brief résumé of the above-mentioned article would not be resented. The disease was first described by Crohn and his associates in 1932, since when only 182 cases have been reported in the United States of America. The condition is usually diagnosed as acute appendicitis. In the differential diagnosis the following have to be considered: (i) mesenteric lymphadenitis, (ii) ileo-caecal tuberculosis, (iii) intestinal obstruction, (iv) ulcerative colitis, (v) neoplasm, (vi) actinomycosis.

The condition is more common in young adults; but the age of persons affected varies from sixteen to fifty-six years. The ætiology is unknown. Crohn grouped the cases into four clinical types representing progressive stages: Group I, in which the symptoms simulate those of acute intraabdominal inflammation, appendicitis in particular; operation reveals a greatly thickened and reddened terminal part of the ileum, which has a tendency to bleed; the mesentery is œdematous, with large, hyperplastic glands. (The case reported here belongs to this group.) Group II, in which the symptoms suggestive of ulcerative colitis occur. Group III, a stenotic stage, following the ulcerative phase as a result of the extreme thickening of the intestinal wall, the lumen gradually becoming constricted. Group IV, which is characterized by the formation of multiple fistulæ, which may open either internally or externally through the abdominal wall. Röntgenological examination may reveal these fistulæ. They persist, and resist surgical measures at closure, unless the bowel is resected.

History and Clinical Findings.

The patient in the case reported here was a well-built girl, aged seventeen years, who was admitted to hospital with a history that three months previously she had had a mild pain under the right costal margin, aggravated by movement or deep inspiration. This pain had occurred intermittently, with no other symptoms, until three weeks before admission, when it became rather worse, and was associated with slight nausea and lassitude. The pain also began to radiate to the left upper part of the abdomen, round beneath the right costal margin, and, on the patient's lying down, into both shoulders; backache also occurred. Fourteen days prior to her admission her private doctor was called and found her suffering from malaise, pain in the right side of the abdomen and vomiting. The next day the doctor visited her again, and found her quite well and free from symptoms. Two days before her admission pain occurred in the right iliac fossa, associated with severe nausea and, later, vomiting; the pain kept her awake most of the night. The medical attendant was called again, and found that the pain had become more severe all over the abdomen, maximal in the right iliac fossa, but there was no rigidity; vomiting had increased. On the morning of her admission to hospital, sixteen hours after his previous visit, he found the patient with a temperature of 37·7° C. (99·8° F.), a rapid pulse, severe pain, tenderness and rigidity in the right iliac fossa, and persistent vomiting. The bowels had functioned regularly; there had been no diarrhœa, and the urinary history was clear. The menstrual history was normal, except that she had had no dysmenorrhœa until her last menses, three weeks previously, when she had had pain in the lower part of the abdomen and backache.

On her arrival at hospital, examination revealed a young girl with a thickly furred tongue and "abdominal" breath. Her temperature was 36·9° C. (98·4° F.), her pulse rate 100 and respiration rate 20 per minute. There were two points at which tenderness was greater than elsewhere: (i) McBurney's point, (ii) one inch to the left of and above the umbilicus. There was general resistance of the abdominal muscles, most pronounced in the right iliac fossa, and the abdomen appeared to be slightly distended. Rosving's sign was positive; but there was no tenderness on rectal examination. The diagnosis of acute appendicitis made by the private practitioner was agreed with, and immediate operation was decided upon.

Operation.

McBurney's incision was made, and when the peritoneum was opened, free blood-stained fluid exuded and inflamed omentum bulged into the wound. A portion of omentum, containing hard material suggestive of thrombi, was later ligated and removed. Distended small bowel was delivered and followed along. At a point 45·0 centimetres (18 inches) from the ileo-caecal junction, a line of inflammatory demarcation occurred, from which point the bowel was less distended. On delivery, the pseudo-obstruction was apparently relieved and the proximal distention subsided. In its last 45·0 centimetres (18 inches), the ileum, except for the final 3·75 centimetres (one and a half inches), was very inflamed, Peyer's patches being swollen to the size of a pea, and to the side of the bowel opposite the mesenteric attachment tags of hæmorrhagic-looking material, similar to that seen in diverticulitis of the colon, were attached. The ileo-caecal area was firmly adherent to the posterior abdominal wall, towards the mid-line, and could not be seen or delivered through the McBurney incision, which was then closed.

A right lower paramedian incision was then made, the ileo-caecal junction brought to view, and the base of the appendix found. The ileo-caecal area was quite normal in appearance. The tip of the appendix was found deeply, and clipped with forceps, and a resolved appendix was removed in a retrograde manner, the stump being invaginated into the caecum.

The uterus, tubes and ovaries were normal. A stab wound was then made in the right iliac fossa, and a quarter-inch drainage tube was inserted into the pelvis. The wound was then closed.

Convalescence.

Following operation, abdominal pain continued for two days, after which the patient was free from it. Drainage had ceased by the second day, when the tube was removed. The patient was given fluids only for two days, then peptonized milk and arrowroot; gradually a light diet was introduced. Her condition continued to

improve rapidly with increasing appetite; but she had no bowel action without an enema up to the time of her discharge from hospital, sixteen days after operation.

Discussion.

Jackson, in his article, agrees that many acute cases must occur in which spontaneous recovery results; but he then goes on to give the views of two schools in regard to surgical treatment when an acute case is encountered. Both schools appear to agree that resection of the inflamed bowel is necessary, but differ as to whether this should be carried out in one stage or multiple stages. Meyer, who is in favour of multiple stages, agrees that if the inflammation is limited to the terminal part of the ileum and the mesentery is uninvolved, resection may not be necessary. It would appear from the result and rapid recovery in this specific case, that it is worth while in the acute case to treat the patient conservatively in the first instance.

Acknowledgements.

In conclusion, I would like to thank Dr. Frank Gill for his permission to report this case, and Dr. Roy Le P. Muecke for his great interest and assistance.

A CASE OF THE ADULT FORM OF STILL'S DISEASE, OR THE STILL-CHAUFFARD SYNDROME.

By C. G. LAMBIE.

(From the Department of Medicine, University of Sydney.)

IN 1897 Still[(1)] described a form of chronic arthritis in children, which he defined as follows: "A chronic progressive enlargement of joints associated with general enlargement of glands and enlargement of the spleen." The onset, he says, is almost always before the second dentition, and is usually insidious, but occasionally it is acute, with pyrexia and perhaps a rigor. Girls are more commonly affected than boys. The joints earliest affected are the knees, the wrists and the joints of the cervical spine, the subsequent order of affection being the ankles, elbows and fingers, while the sterno-clavicular and temporo-maxillary joints are occasionally involved. The affection is symmetrical and there is no tendency to suppuration or to bony ankylosis. The muscles activating the diseased joints show early and marked wasting. The glands most commonly affected are the supratrochlear, the axillary, the inguinal, and those in the posterior triangle of the neck. The enlargement of the spleen and glands is proportional to the severity of the condition. Anæmia and attacks of pyrexia are also features of the disease. At *post mortem* examination the joints show thickening of the capsule and connective tissue just outside them, also thickening and vascularization of the synovial membrane and fibrous adhesions. There is, however, a complete absence of lipping or destruction of cartilage.

It is evident from the above description that Still's disease closely resembles rheumatoid arthritis in adults, with the addition of glandular and splenic enlargement.

In 1896, the year preceding the publication (but subsequent to the writing) of Still's paper, Chauffard and Ramond[(2)] described an acute form of rheumatoid arthritis, referred to by them as "infective arthritis", which was accompanied by enlargement of lymphatic glands; but they made no mention of enlargement of the spleen. Similar cases were also reported by Bannatyne and Wohlmann,[(3)] of Bath (1896), who, in addition to glandular enlargement, deforming arthritis and anæmia, mentioned skin pigmentation as a feature of the disease.

In 1904 McCrae,[(4)] in reviewing 110 cases of *arthritis deformans* (including two cases of Still's disease) in Osler's clinic at the Johns Hopkins Hospital, mentioned the occurrence of general glandular enlargements in thirteen, of enlargement of the spleen in four, of pigmentation in eight, and of leucopenia in six cases. Since then numerous Continental writers have reported cases of the adult type of Still's disease, usually under the title of the Still-Chauffard syndrome; but the disease seems to have been overlooked by writers in English-speaking countries until Felty[(5)] in 1924 described a series of cases and emphasized the main characteristics of the syndrome, namely, deforming arthritis, enlargement of lymphatic glands and spleen, anæmia, leucopenia and pigmentation of the skin. Since the appearance of Felty's paper the syndrome has been referred to by American authors as Felty's syndrome.

In addition to pigmentation, skin rashes constitute an occasional feature of both the juvenile and the adult form of Still's disease. Various types of rashes have been reported: urticaria, *erythema multiforme* (Bailey and discussion by Parkes-Weber, 1934), pemphigus (Graber-Duvernay and Gerbay, 1934[(7)]).

As regards the ætiology of the condition, the suggestion that it is of tuberculous origin (Edsall, 1904,[(8)] Parkes-Weber, 1905,[(9)] Piske, 1913,[(11)] Longo, 1917[(16)]) or of syphilitic origin (Weissenbach and Françon, 1931[(17)]) may be discounted as being unsupported by adequate evidence. Within recent years evidence has been accumulating which points to the condition as being a very chronic form of *sepsis lenta*. In 1923 Iseke[(13)] obtained at *post mortem* examination pure cultures of a streptococcus of the *viridans* type from the spleen, lungs, endocardium and middle ear; but until 1926, when Reimold and Stöber[(12)] cultivated *Streptococcus viridans* from the blood of a child during life, attempts to grow organisms from the blood proved vain. *Streptococcus viridans* has also been isolated from the blood in the adult form of Still's disease, and Debré, Broca and Lamy (1930),[(6)] and Singer and Levy (1936)[(14)] have recorded cases in which the syndrome was associated with typical bacterial endocarditis. A constant relationship between chronic infections with streptococci of the *viridans* type and the Still-Chauffard syndrome cannot yet, however, be regarded as having been established.

As regards treatment, splenectomy and the administration of gold salts (Graber-Duvernay and Gerbay, 1934[(7)]) have been used, and good results are reported from this treatment; but the number of cases studied is too small to justify the drawing of any general conclusions.

Up to the present no case of the Still-Chauffard syndrome seems to have been reported in Australia. It is therefore of interest to place the following case upon record.

Case History.

I.P., a married woman, aged fifty years, was admitted to the Royal Prince Alfred Hospital on October 28, 1935, complaining of inability to walk or to use the hands freely for two years, and of itching all over the body of three months' duration.

She gave the history that she was in good health until January, 1928, when, just after the birth of the second of her two children, she developed pain and swelling of most of her joints. The pain began in the ankle joints and moved from joint to joint. She was treated for rheumatism, but was never put to bed at any time. In June, 1928, some deformity of the hands began to appear. Later, the elbows, feet, knees and left hip joint became affected. In 1933 she began to complain of pain and stiffness in the neck and back on sitting up for more than a short time. By this time she also had such difficulty in getting about that she had to take to crutches.

In August, 1935, she had an attack of urticaria, and between then and her admission to hospital in October of that year she had two or three attacks almost daily. There were no symptoms referable to the cardio-vascular, respiratory, urinary or alimentary systems.

As regards her previous health, she had had tonsillitis and muscular rheumatism in 1912 and jaundice in 1924. All her teeth were removed in March, 1935.

Nothing relevant was elicited from the family history. No member of the family had suffered from rheumatic or cardiac disease. She had two children, both of whom were in good health. The patient was engaged in domestic duties and never had any but light work to do. Her diet

appeared to have been adequate, and she was a total abstainer.

On physical examination the patient was seen to be emaciated, pale and weak. The knuckles were swollen, and flexion and ulnar deviation of the fingers were present at the metacarpo-phalangeal joints. The terminal phalanges were flexed. These deformities are shown in Figure I. The wrist joints were swollen and slightly flexed. Movement of all the joints of the hand was limited, and crepitus could be elicited at the wrist joints. The small muscles of the hands, with the exception of those of the hypothenar eminences, were wasted. The elbow joints were maintained in a position of partial flexion, and there was limitation of extension at these joints. The feet showed *talipes equino-varus* with hyperextension of the toes, except the great toes, which were flexed at the metacarpo-phalangeal joint and extended at the terminal interphalangeal joint. The ankle joints were swollen. There was little, if any, limitation of movement at these joints. The muscles of the feet and legs, like those of the hands and forearms, were wasted. The knee joints were swollen, but not painful, and marked crepitus could be elicited at them. The hip and shoulder joints appeared to be unaffected. The spine was rigid.

FIGURE I.

Showing deformities of hands due to arthritis in a case of the adult form of Still's disease.

X ray examination of the joints revealed the presence of great osteoarthritic changes involving the distal ends of both radii and ulnæ and of all the carpal bones, as well as complete destruction of both radio-carpal joints. These changes were most pronounced on the left side, where the carpal bones appeared to be dislocated slightly forwards toward the palmar aspect of the hand.

The spleen was enlarged, the lower border being palpable three fingers' breadth below the costal margin.

Three enlarged lymphatic glands, each about 1·25 centimetres (half an inch) in diameter, were palpable in the right axilla.

The heart was somewhat enlarged, the apex beat being palpable 1·25 centimetres (half an inch) outside the mid-clavicular line in the fifth left intercostal space. The left border of deep cardiac dulness was encountered in this interspace 10·0 centimetres (four inches) to the left of the middle line. The right border could not be delimited. A soft systolic murmur was present, having its maximum intensity in the mitral area and propagated towards the axilla.

The pulse rate was 88 per minute; the systolic blood pressure was 130 millimetres of mercury and the diastolic 98 millimetres of mercury. No Osler's nodes or skin rashes were present. The temperature was normal.

Examination of the blood revealed the presence of leucopenia and anæmia. The leucocytes numbered 4,500 per cubic millimetre, 60% being neutrophile cells, 30% lymphocytes, 2% monocytes and 8% eosinophile cells. The erythrocytes numbered 3,700,000 per cubic millimetre. The hæmoglobin value was 69%. The Schilling count revealed an increase in the number of band forms (19). No organisms were grown on attempted culture from the blood.

The blood uric acid content was three milligrammes and the blood sugar content 79 milligrammes per 100 cubic centimetres of blood. The basal metabolic rate was +8%.

No reaction was obtained to the Mantoux test, and X ray examination of the chest revealed no evidence of pulmonary tuberculosis or other pulmonary disease.

The urine contained a trace of albumin, but no blood corpuscles, casts or other abnormal constituents. The curve of gastric acidity was normal.

The patient had almost daily attacks of urticaria throughout her stay in hospital.

She was readmitted to hospital in October, 1937. The condition of her joints had remained much the same; the spleen was also still palpable about the same distance below the costal margin. The glandular enlargement and the anæmia had, however, disappeared. The systolic murmur in the mitral area was no longer audible, but there was a persistent reduplication of the first sound. The heart was still enlarged and the electrocardiogram showed evidence of intraventricular block and of occasional ventricular extrasystoles. The leucocyte count was now slightly higher, but it remained slightly below normal, namely, 5,100 per cubic millimetre. The sedimentation rate was greatly increased. The blood was still found to be sterile.

Comment.

It will be seen from the above description that the case resembled the recorded cases of the Still-Chauffard or Felty's syndrome in respect of the following features: arthritis, splenic and glandular enlargement, urticaria, leucopenia and anæmia. The condition differs from the classical description of Still's disease, however, in that osteoarthritic changes had supervened. It is doubtful whether this difference is a sufficient reason for regarding the condition as essentially different from Still's disease, since an arthritis of the osteoarthritic type may be secondary to one of the rheumatoid type.

Still's disease should be classified as a variety of chronic infective arthritis. The condition present in the case under discussion would correspond to the third stage in the evolution of chronic infective arthritis, namely, that in which occur loss of cartilage and subsequent erosion of articular bones, followed by subluxation, and disorganization of the joints with fibrous and bony ankylosis, more especially of the small joints of the hand.[(a)]

The changes in the spleen, in the lymphatic glands and in the heart would seem to suggest that in Still's disease the organisms supposedly responsible for the condition more frequently enter the blood and tissues than in the common forms of chronic rheumatoid arthritis. The destructive changes in the joints in the present case would likewise point in the same direction. This view receives support from the reports of those observers who have isolated green streptococci from the blood in similar cases. In the case here reported, however, the blood was consistently sterile.

References.

(a) R. H. Bailey: "Still's Disease with Erythema Multiforme", also discussion by Parkes-Weber, *Proceedings of the Royal Society of Medicine*, Volume XXVIII, 1934, Section of Pædiatrics, page 157.

(b) G. A. Bannatyne and A. S. Wohlmann: "Rheumatoid Arthritis: Its Clinical History, Ætiology and Treatment", *The Lancet*, Volume I, 1896, page 1120.

(c) A. Chauffard and F. Ramond: *"Des adénopathies dans le rheumatisme chronique infectieux"*, *Revue de médecine*, Volume XVI, 1896, page 345.

(d) R. Debré, R. Broca and M. Lamy: *"Forme endocardique de la maladie de Still"*, *Archives de médecine des enfants*, Volume XXXIII, 1930, page 212.

(e) D. L. Edsall: "Concerning the Nature of Still's Type of Chronic Polyarthritis in Childhood", *Archives of Pediatrics*, Volume XXI, 1904, page 175.

(46) A. R. Felty: "Chronic Arthritis in the Adult Associated with Splenomegaly and Leucopenia: A Report of Five Cases of an Unusual Clinical Syndrome", *The Bulletin of the Johns Hopkins Hospital*, Volume XXXV, 1924, page 16.
(47) J. Graber-Duvernay and F. Gerbay: *"Une nouvelle observation du syndrome de Chauffard-Still de l'adulte: Les réflexions qu'elle suggère"*, *Lyon médical*, Volume CLIX, 1934, page 589.
(48) G. Iseke: *"Zur Ätiologie der Stillschen Krankheit"*, *Zeitschrift für Kinderheilkunde*, Volume XXXV, 1923, page 315.
(49) A. Longo: *"Contributo allo studio della così detta malattia di Still"*, *Rivista di clinica pediatrica*, Volume XV, 1917, page 225.
(50) T. McCrae: "Arthritis Deformans: The Report on 110 Cases from the Johns Hopkins Hospital", *The Journal of the American Medical Association*, Volume XLII, 1904, page 1.
(51) J. Piske: *"Zur Kenntnis der Stillschen Krankheit"*, *Medizinische Klinik*, Volume IX, 1913, page 1968.
(52) W. Reimold and T. Stöber: *"Beiträge zum Problem der Stillschen Krankheit"*, *Monatsschrift für Kinderheilkunde*, Volume XXXI, 1926, page 597.
(53) "Reports on Chronic Rheumatic Diseases: Annual Report of the British Committee on Chronic Rheumatic Diseases Appointed by the Royal College of Physicians", Number 1, 1935, pages 18 and 32; *ibidem*, Number 2, 1936, page 1.
(54) H. A. Singer and H. A. Levy: "Relationship of Felty's and Allied Syndromes to Sepsis Lenta", *Archives of Internal Medicine*, Volume LVII, 1936, page 576.
(55) G. F. Still: "On a Form of Chronic Joint Disease in Children", *Medico-Chirurgical Transactions*, Volume LXXX, 1897, page 47.
(56) F. Parkes-Weber: "Still's Type of Chronic Joint Disease in Children and the So-Called Tuberculous Rheumatism", *The British Journal of Children's Diseases*, Volume II, 1905, page 208.
(57) R. J. Weissenbach and F. Françon: *"Le syndrome de Chauffard-Still; rhumatisme chronique fibreux déformant progressif avec adénopathies et splénomégalie: Sa place en nosologie"*, *La presse médicale*, Volume XXXIX, 1931, page 1197.

Reviews.

BIOLOGY FOR MEDICAL STUDENTS.

A NEW edition of an introductory work such as "Biology for Medical Students", by Hentschel and Cook,[1] tempts us to take a wider view and to glance at the need for such a book and at the group of students demanding it.

In recent years, with the ever-increasing volume of research and discovery in medicine, the medical student has been overwhelmed with the mass of knowledge presented to him. His teachers cry out aloud for more time, whilst exponents of new subjects proclaim the importance of their stock-in-trade. We believe that the future will have to bring some better programme than we have at present.

Notwithstanding criticism of the preclinical years there is much evidence to show that a fundamental scientific understanding is essential for the later years of medical study, and anyone conversant with the almost complete ignorance of the "life sciences" exhibited by the first-year medical student will find an important service in the zoology and botany courses, which together make up the content of biology. It would, however, be fair to expect the programmes of the premedical sciences to be formulated with the closest view to the ultimate aims of the student, provided that this does not clash with other and equally deserving viewpoints.

Most biology text-books for the first-year student cover the zoology or botany sections alone. A single volume covering the whole of biology might be very desirable. The present book is supposed to supply this want. Unfortunately the volume under review, whilst excellent in many respects, is still made up of two separate subjects bound up in the same cover. Probably that will always be the result when two authors write different sections.

Into the second edition the authors have introduced additional types in view of the requirements of certain English universities. It is very questionable whether their choice of types is the most suitable, but since the authors inform us that they have deliberately chosen to make the book cover the new syllabuses of Oxford and Cambridge, this choice is not exactly theirs. Still, to us, it seems curious that, notwithstanding the devotion of a whole chapter to the fungi with detailed life histories of several of them, there is no reference to *Tinea* or other fungi causing human disease. And this is only one example of the kind.

Our chief criticism of the book is that the words of the title, "for Medical Students", might in its application to many sections be left out. On the other hand the book is beautifully illustrated and the general production is excellent. The details appear to be up to date and a few errors of the first edition have been eliminated.

[1] "Biology for Medical Students", by C. C. Hentschel, M.Sc., and W. R. I. Cook, B.Sc., Ph.D., with a foreword by G. E. Gask, C.M.G., D.S.O., F.R.C.S.; 1937. London: Longmans, Green and Company. Demy 8vo, pp. 676, with illustrations. Price: 18s. net.

"THE COMPLEAT PEDIATRICIAN."

WE have just received the second completely revised and rewritten edition of "The Compleat Pediatrician".[1] Three years ago we reviewed the first edition of this carefully indexed pædiatric "omnibus" and outlined the principles on which the work was based. The author now defines three hundred and twenty-nine separate pædiatric diseases, of which approximately "only one hundred are important", since these can either be prevented or will respond to adequate therapy, while children suffering from the other two hundred and twenty-nine usually recover or succumb regardless of therapy.

Dr. Davison assumes that with the assistance of this medical lexicon every family physician, aided by "the patient's pain, appearance, neuro-psychiatric symptoms and the information obtained by palpation, percussion, auscultation and laboratory tests, can diagnose and treat successfully" the greater majority of these curable and preventible diseases. Nevertheless, we may express grave doubt of the utility of the following method of approach to an everyday problem:

> For example, if the patient's chief complaint is headache, the physician should consult the paragraph on headaches. . . . He should concentrate on the more likely possibilities, some of which he probably had overlooked. He especially should consider the diseases for which adequate therapy is available, e.g., the causes of headache listed in paragraph ninety-two include meningococcus meningitis, scarlet fever, sinusitis, eye strain, etc. The reference numbers after each disease and symptom indicate paragraphs in which attention is called to additional symptoms and signs which should be present for a diagnosis, to other diseases which will cause similar symptoms, to various diagnostic laboratory methods, lumbar puncture, spinal fluid examination, the Schultz-Charlton blanching test, x-ray and ophthalmoscopic examinations, to the most useful therapeutic procedures, for example, anti-meningococcus or anti-scarlatinal serum, sulfanilamide, ephedrine sprays, and the correction of refractive errors, and any possible preventive measures.

Despite its many peculiarities this new edition contains an enormous amount of carefully arranged pædiatric information. The main text, which in the first edition consisted of numbered paragraphs, has been subdivided into seven chapters "on the basis of the anatomical system chiefly involved". New chapters on growth, development and nutrition have been added.

[1] "The Compleat Pediatrician: Practical Diagnostic, Therapeutic and Preventive Pediatrics", by W. C. Davison, M.A., D.Sc., M.D.; Second Edition, completely rewritten; 1938. Durham: Duke University Press. Royal 8vo, pp. 250. Price: $3.75 net.

The Medical Journal of Australia

SATURDAY, JUNE 25, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

BABEL.

FROM time immemorial man has sought the elixir of life, hoping thereby to prolong his earthly existence and even the life beyond the grave. Osler sounded the note of partial success in his review of the advance of medical knowledge up to the year 1901: "For countless generations the prophets and kings of humanity have desired to see the things which men have seen, and to hear the things which men have heard in the course of the wonderful nineteenth century. . . . Measure as we may the progress of the world materially, in the advantages of steam, electricity and other mechanical appliances; sociologically, in the great improvement in the conditions of life; intellectually, in the diffusion of education; morally, in a possibly higher standard of ethics—there is no one measure which can compare with the decrease of physical suffering in man, woman, and child when stricken by disease or accident. This is the Promethean gift of the century to man."

Less than forty years have passed since Osler wrote, and yet it may now be argued with considerable force that all these great advances in knowledge have recoiled on man, so that, far from being a blessing, they are a curse. The prolongation of life has occurred so suddenly in the history of mankind that the attendant ills seem to be greater than the benefits. When the history of this period comes to be written in its true perspective the social confusion of the present day, with men's hearts failing them for fear, will be attributed to deeper and more significant forces than the Great War.

The social life of the community has failed to adjust itself to the increased expectation of life of the individual. In a large majority of articles written on labour conditions, on university education, on economics and on other subjects, the effects of the Great War are prominently displayed; but a careful analysis discloses a failure on the part of the authors to appreciate the significant influence of the increased span of life on these activities of man.

The lower animals soon cast off parental care and bodily maturity coincides fairly closely with sexual maturity. In man sexual maturity is as it were premature, and the more the period of adolescence is lengthened by social conditions, the greater becomes the confusion. The editor of the Concise Oxford Dictionary defines adolescence as the period of "growing up, between childhood and manhood (14 to 25) or womanhood (14 to 21)"—truly an old man's definition. It may be significant that he gives no years in the definition of senile ("showing the feebleness &c. of old age"), but it is generally agreed that the advent of senility today has been considerably deferred by the higher standards of public health. Gradually, by the efflux of time, provided no embarrassing initiative is shown, those in pensionable occupations are confident of reaching their retiring age with health unimpaired. If they are not in pensionable occupations, the boon of health allows them to continue accumulating wealth in the full vanity of feeble old age. Such is the Promethean gift to humanity. But always pushing upwards and storming the heavily fortified citadels of old age are the implacable forces of youth.

Up to the present, youth has failed to obtain due recognition. The repeated suggestions from different parts of the world that the school leaving age should be increased, the recent variation of the Federal awards in relationship to apprenticeship, the increasing age at which students are becoming qualified in the various professions, the whole

problem of the employment of women, and many other social problems are in a very large measure expressions of the fact that the age periods of life are becoming extended. Unfortunately the advent of sexual maturity has not been advanced to coincide with the arbitrarily chosen moment when adolescence ceases, and hence the marriage state is not entered upon at a stage early enough to establish that family bond of union which is the foundation of the State. The adolescent of both sexes hurries from one excitement to another in search of satisfaction for those emotional stimuli which he or she so little understands. Large numbers of young women are for ever debarred from maternity and sublimate their instincts in various forms of social service. Much of this social service is given without previous or concurrent study of those factors which have created the need for the service. The social confusion caused by the interference with a biological equilibrium cannot be calmed without reference to the ætiological factors which have brought it about.

At the other end of the time scale the effects of the improvement in the public health are most vividly shown in the age of the majority of those who are found in places of rule. In passing it may be remarked how little the profession which is responsible for this prolongation of life is represented in the affairs of government.

Current Comment.

ALKALIS AND PLEONEXY.

In view of the enormous quantity of alkalis administered to patients suffering from gastric and duodenal ulceration and diseases complicated by ketosis or acidosis, an authoritative pronouncement would be welcome as to the possibilities and dangers of alkalosis. J. K. Latchford remarks that although some physicians are aware of the dangers of sodium bicarbonate, it is desirable that the findings of J. Barcroft regarding the pleonectic tendency of sodium bicarbonate should be more widely appreciated.[1] It might then be clearly recognized that there is danger in the careless employment of sodium bicarbonate, which may exert its anoxigenic action or capacity to produce anoxæmia (or anoxia), the result depending on the dose administered and the resistance of the individual to the tissue breakdown due to oxygen deprivation. Latchford had his attention directed by two happenings to the misplaced trust reposed in sodium bicarbonate for the relief of gastric distress. A man, seventy years of age, consumed during one night large quantities of baking soda dissolved in water, to relieve epigastric distress. As a result of the accumulation of carbon dioxide gas, with coincident pylorospasm and cardiospasm, his stomach became greatly distended and his respirations were much diminished in volume. Relaxation of the pylorus and cardia was effected and the gas evacuated through the natural channels, the intragastric tension and consequent epigastric pain being thereby alleviated. Another patient employed bicarbonate of soda for the relief of epigastric pain. This treatment resulted in the patient's vomiting and experiencing a faint feeling for some days after the last attack of pain. He had been taking the bicarbonate for some months. In these and other instances bicarbonate of soda taken alone or with other carbonates has led to a definite sequence of events. Discomfort or pain leads to the taking of soda (often in teaspoonful doses), which is followed by sensations of weakness or faintness or by actual collapse. It is not uncommon to learn that, after the taking of soda, collapse or even sudden death of a person supervenes. It is possible to state definitely that in some cases the bicarbonate, far from relaxing the cardia or pylorus, merely aggravates the gastric distension and causes the physician to make the erroneous diagnosis of *angina pectoris*. Instances of tetany have been reported and ascribed to the use of bicarbonate of soda.

Barcroft points out that sodium bicarbonate, like carbon monoxide, interferes with the discharge of oxygen by the circulating hæmoglobin. In this fact lies all the danger of sodium bicarbonate. Barcroft has also demonstrated that this substance is highly pleonectic in its action on hæmoglobin. He points out that pleonexy (erroneously spelt "pleionexy" in the text) is that condition in which the circulating hæmoglobin holds more firmly to its gathered oxygen than in the normal state (mesonexy). Accordingly it gives off a less than normal amount of oxygen to the tissues. When pleonexy exists, whether induced by sodium bicarbonate, dibasic sodium phosphate or diminished carbon dioxide content, the tissues get insufficient oxygen for their requirements and fail in proportion to their oxygen lack. With the onset of pleonexy an anoxæmic condition supervenes and the organism may succumb. The effect of sodium bicarbonate in inducing pleonexy is proportional to the quantity absorbed and the ability of the organism to withstand the anoxæmia thus induced.

Not all degrees of anoxæmia are fraught with danger, but they all tend to cause tissue breakdown. This action is irrespective of whether the anoxæmia is due to rarefied altitude, strenuous muscular exercise or diminution of the oxygen-carrying power of the blood following severe hæmorrhage. When

[1] *The Canadian Medical Association Journal*, April, 1938.

the person's tissues cannot withstand the anoxæmic breakdown, failure of function is inevitable. Latchford states that sodium bicarbonate shifts the dissociation curve of hæmoglobin to the left, decreasing the oxygen supply to the tissues and inducing pleonexy. There can be no justification for allowing this condition to be produced by therapeutic measures, for immediate interference with oxygen supply follows absorption of the bicarbonate.

J. J. R. MacLeod, in his study by animal experiments of lactic acid concentrations in conditions of anoxæmia and shock, found that ingestion of sodium bicarbonate with food was followed by an increase of the concentration of lactic acid in the blood and urine. Increased lactic acid content has been accepted as a definite indication of the anoxæmic state. MacLeod confirmed Barcroft's observations by showing that anoxæmia may follow the administration of sodium bicarbonate. Blatchford points out that suddenly fatal anoxæmia may follow the administration of a large dose of bicarbonate. C. S. McVicar remarked on the high colour of the lips and cheeks in patients suffering from gastric tetany, a condition which he considered was probably due to the high percentage of hæmoglobin in the blood and the decreased vasomotor tone of the blood vessels. But Latchford suggests that this condition might also be explained by a high saturation of hæmoglobin with oxygen. This feature of the pleonectic state is often attributed to acidosis. The blood holds its oxygen. The normal production of carbon dioxide is diminished. Cyanosis may be absent even at the time of actual failure.

Latchford's article contains much that is of great interest. It discloses unsuspected perils in such a commonplace and familiar remedy as baking soda. Further, it makes us realize how much safer is the use of oxide of magnesium when the administration of an alkali over long periods is indicated.

THE ADMINISTRATION OF THYREOID GLAND TO PREMATURE BABIES.

THE blood of new-born babies contains a comparatively large amount of œstrin, which is rapidly excreted by the kidneys and ceases to be present in the urine about four days after birth. The contention has been made that a baby born prematurely lacks the stimulus of the œstrin which would have continued to abound in its blood until the full term of pregnancy had the fœtus remained *in utero*. Some authorities have maintained that the absence of this stimulus may explain the general depression of metabolism which constitutes one of the major handicaps of the premature baby during the early days of life. In 1936 Alan Moncrieff observed the effect of subcutaneous injections of œstrin on premature babies during the first seven days of life, and concluded that the advantages produced did not appear definite enough to warrant the routine administration of this substance to premature babies.

It would appear that the administration of thyreoid gland substance to premature babies was first suggested by Pritchard in 1934. This form of treatment is based on Gudernatsch's work in the years 1911 to 1914, on the feeding of tadpoles with thyreoid gland, and his conclusion that "the mammalian thyreoid contains an *agens* which, when given as food, calls forth a rapid differentiation in a developing vertebrate organism". Gudernatsch emphasized that it was differentiation of function and structure, and not growth, which was achieved in the tadpole.

Alan Moncrieff has more recently reported the results of a very carefully controlled investigation of the effect of the administration of thyreoid gland to premature infants during the first two weeks of life.[1] This study was carried out in the wards of the Queen Charlotte's Maternity Hospital and was based on the observation of sixty-two babies weighing five pounds or less at birth. Alternate babies received thyreoid gland contained in lactose tablets or plain "dummy" tablets. The daily dosage used was one-twentieth of a grain of thyreoid gland for each pound of body weight for the first two weeks of life. The gland substance was administered to these babies, not with the object of supplying a deficiency, but in the hope that it would hasten maturation and remove the inertia which makes the premature baby so difficult to manage.

During the course of this investigation the opinion was frequently expressed by the nurses in charge that the "treated" babies were really easier to manage, and it soon became impossible to conceal the fact that the "dummy" tablets did not contain any thyreoid gland, for the babies taking them were thought by the nursing staff to be more difficult to feed and to keep warm than those who were receiving the gland.

Obviously any striking effect on the weight of the babies receiving thyreoid gland was not to be expected, since its stimulating action on appetite and digestion might well be counterbalanced by the general raising of the metabolic rate. The effect on weight may be summarized by the statement that the drug administered in the dosage employed in this investigation did not produce any obviously beneficial change in the weight curve. Rectal temperatures were taken night and morning. Although the untreated babies appeared to have a higher "lowest temperature level", the emphatic opinion of the nursing staff was that the babies receiving the gland were more easily kept warm.

The contrast in the death rate for these two groups was very marked, the mortality rate being 6% in those receiving thyreoid gland, and 17% in those not treated. As the author points out, the figures are small, but since all the conditions of nursing, care and feeding were identical and were carried out by the same staff during the same period of time, the difference in the mortality rates may be regarded as significant and represents the main argument in favour of thyreoid gland administration.

[1] *Archives of Disease in Childhood*, Volume XIII, March, 1938, page 57.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on March 31, 1938, at the Anatomy Theatre, University of Adelaide, DR. R. E. MAGAREY, the President, in the chair.

Cerebral Vascular Accidents.

DR. C. T. CH. DE CRESPIGNY read a paper entitled "Cerebral Vascular Accidents and Their Treatment" (see page 1077).

DR. H. K. FRY said that opinions in regard to cerebral vascular accidents had changed radically in the past twenty years. *Pachymeningitis hæmorrhagica interna*, when he was a student, was considered to be the result of an obscure local inflammation of the dura, as Virchow had taught. Bleeding from the thin-walled cerebral veins was at the present time the accepted explanation. Sperling had demonstrated that the subdural injection of blood led to typical pachymeningitis, while a similar injection of defibrinated blood did not. Wood Jones had pointed out that epidural hæmatoma was usually due to venous, not to arterial bleeding. The congenital berry aneurysms had been given due recognition only in recent years, while Charcot's miliary aneurysms had been shown to be small dissecting aneurysms, more often the result than the cause of hæmorrhage. Abbie had denied the existence of Duret's lenticulo-optic artery recently, but the description of the lenticulo-optic artery in the Sydenham Society's edition of Charcot's works corresponded approximately to Abbie's deep optic branch of the lateral striate group. With regard to treatment, the most important procedure was to relieve the cerebral anæmia or anoxæmia produced by an increase in volume of the extravascular contents of the rigidly enclosed cranial cavity. Epidural and subdural clots presented a purely surgical problem. Subarachnoid hæmorrhage demanded repeated drainage by lumbar puncture. The diminution of extravascular pressure might be thought to be provocative of increased bleeding, but the fact that increased intracranial pressure led to an increased systemic blood pressure was of more importance. The diagnosis of intracerebral hæmorrhage from cerebral thrombosis was often difficult and even impossible. Thrombosis was probably more common than hæmorrhage. Collier's teaching that stimulating treatment should be given was most rational, unless the evidence of hæmorrhage was patent. After the apoplectic period the use of the time-honoured combination of potassium iodide and aromatic spirits of ammonia was reasonable. A starvation diet would not cure, but might augment atheromatous changes, and would predispose to thrombosis. A reduced but well-balanced diet was indicated.

DR. IAN HAMILTON inquired what was the shortest time required for death to occur from a spontaneous intracranial hæmorrhage. He had seen a young married woman, about twenty-five years of age, who was in the habit of going motoring at night with two men to a lonely seaside place. She had gone on a number of occasions, but on the last occasion, whilst engaged in some sort of scuffle with the men she had suddenly collapsed and was dead apparently within a few minutes. Her companions drove her to a doctor and the police were informed. Apparently there was no question of foul play in the case. On performing a *post mortem* examination Dr. Hamilton had discovered a large subarachnoid blood clot, some inches in extent, at the base of the brain, which had been caused by a rupture of a very small aneurysm of the basilar artery. The interpretation of the case seemed to be that her blood pressure had risen somewhat during her struggles with her friends, and her aneurysm had burst. This had caused a rapid increase of intracranial pressure, which had given rise to a very sudden loss of consciousness; but the size of the blood clot indicated that death could not have taken place at the very least for five to fifteen minutes, possibly more.

A meeting of the New South Wales Branch of the British Medical Association was held on April 14, 1938, at the Royal Alexandra Hospital for Children. The meeting took the form of a number of clinical demonstrations by members of the honorary staff. Part of this report appeared in the issue of June 18, 1938.

Coarctation of the Aorta.

DR. R. J. TAYLOR showed a girl, aged eight years, who had previously suffered from pertussis, measles and pneumonia. Breathlessness and palpitation had developed after pneumonia, which the child had eighteen months previously. She did not go blue and had no cough.

On examination a mild degree of cardiac enlargement was found. There was a harsh aortic systolic murmur, propagated into the vessels of the neck. An accentuated aortic second sound was heard. A large artery was found running down superficial to the right scapula. There was an absence of pulsation in the line of femoral vessels. The systolic blood pressure in the right arm was 134 and in the left arm 138 millimetres of mercury; the diastolic blood pressure was 90 millimetres of mercury. No sounds were heard in the popliteal fossæ.

X ray examination on March 28, 1938, revealed hypertrophy of the left ventricle, absence of the aortic knob and some irregularity of the under-aspect of the ribs, as well as some dilatation of the ascending arch of the aorta. The findings suggested coarctation of the aorta.

Retroperitoneal Sarcoma.

DR. W. VICKERS showed a girl, aged nine years, who had previously suffered from scarlet fever, whooping cough and measles. The family history was clear. The child had been limping on the right leg for three months; the limp had been gradually becoming worse, and was accompanied by severe pain in the back. The child was losing weight. There was no history of injury.

Physical examination revealed a well-nourished child, who walked with an obvious limp of the right leg. No shortening and no wasting of the thigh or the leg had occurred. All movements of the right hip joint were slightly limited and painful. No response was elicited to the von Pirquet test. X ray examination of the hips revealed no abnormality of the sacro-iliac joints or of the spine. Examination of the blood revealed the presence of a secondary anæmia.

The child was discharged and was readmitted three weeks later with very severe pain in both legs; she was screaming with pain and complaining of numbness in the gluteal region and in the right foot.

On examination a swelling was found in the right iliac fossa and the right loin. A large, hard, well-defined mass occupied the whole of the right iliac fossa; it was tender on pressure, and muscular rigidity was present. Laparotomy was performed and revealed a retroperitoneal sarcoma in the right side of the abdomen. Retention of urine developed after the operation, together with loss of sensation over the dorsum of the right foot and half way up the calf of the right leg, and loss of dorsiflexion and evertion of the right foot. The child was given deep X ray therapy and gradually improved. At the time of the meeting she still had some incontinence of urine, but had control of her bowels.

Residual Paralysis.

Dr. Vickers next showed a girl, aged two years, who had been admitted to hospital on October 22, 1934. She had previously suffered from bronchitis. She had been ill for two weeks prior to admission. She was listless, drowsy and feverish, and resented being sat up or moved. No neck rigidity was present, but the child seemed to have some difficulty and pain with micturition. The bowels were well open and the child was taking food.

On examination she appeared listless and feverish; the tongue was furred and the tonsils were slightly reddened. No abnormality was detected in the abdomen or in the respiratory or cardio-vascular system.

Examination of the nervous system revealed no spine sign, no Kernig's sign and no neck stiffness. The knee jerk was exaggerated and the abdominal reflexes were normal. The child did not appear to be in any way paralysed, but she did not breathe with the intercostal muscles.

On October 29, 1934, the intercostal muscles were very weak; the child was passing urine satisfactorily. On November 16, 1934, the muscular weakness was generalized, but paralysis was noted only in the left deltoid and intercostal muscles. Massage treatment was being given. On November 28, 1934, the child's condition was improving, but she was still not using the intercostal muscles. On November 30, 1934, she was discharged from hospital to attend at the massage and follow-up departments.

Reexamined in March, 1938, the child had complete paralysis of the intercostal muscles. She ran about and was not at all distressed.

Scurvy.

DR. D. G. R. VICKERY showed a female infant, aged six months, who had been fed on cow's milk. She had resented movement of the left thigh for one week.

On physical examination her gums appeared slightly red, but not hæmorrhagic. Both thighs were very tender, but no abnormality of contour was detected. The tenderness was not localized. The child lay with the hips internally rotated and slightly abducted; she resented movement of the hips. Some subluxation of the costo-chondral junctions was noted. The cerebro-spinal fluid was clear. X ray examination of the hips suggested fairly pronounced scurvy.

DR. C. L. S. MACINTOSH showed a male baby, aged nine months, who was admitted to hospital on March 13, 1938, on account of not responding to a "special diet". He was a pale, miserable and listless weakling, lying with the knees partly flexed, drawn up and rotated outwards. There were obvious swellings of the left leg and about the right knee joint. Palpation of the lower limbs was resented and the child became irritable. The sole diet had been boiled milk since birth and prior to treatment (one quart daily). The skin was pale and moist. At the time of the child's admission to hospital pronounced sclerodermatous areas were visible on the posterior aspect of the left leg and thigh and extending to the buttocks, also about the right knee joint and the posterior aspect of the right arm. A week later this condition began to subside, and at the time of the meeting it had disappeared.

When the child was admitted to hospital the gums were purplish, swollen, spongy and bleeding. This condition had subsided. The abdomen was distended, but soft, and the liver reached one finger's breadth below the costal margin; the spleen was not palpable. Constipation had been troublesome during the child's stay in hospital, and at times fæcal masses could be felt through the abdominal wall. The stools contained many hard fæcal masses.

The fronto-parietal regions of the skull were prominent, which gave the head a "square-head" appearance. The anterior fontanelle was not completely closed; this was about the average for nine months. Pronounced and visible bilateral swellings, possibly beading, were found at each costo-chondral joint and were probably hæmorrhagic in origin. The lower extremities were tender to the touch, but much less so than a short time previously, especially in the case of the left leg and of both thighs, and in these areas considerable thickening could be felt.

X ray examination on March 14, 1938, revealed advanced scorbutic changes in the lower limbs. In a few days these changes progressed and slight elevation of the periosteum of both femora and of the left tibia occurred. A week later the subperiosteal hæmorrhages had become considerable, and there was evidence of early calcification beneath the raised periosteum; by April 4, 1938, calcification had become much more pronounced and more dense, being very marked in the case of the left tibia.

Examination of the blood revealed 2,600,000 erythrocytes per cubic millimetre. The hæmoglobin value was 26% and the colour index was 0·5. The cells were pale, with wide variation in size and shape. Many microcytes were present, reticulocytes numbered less than 1%, and five normoblasts were seen. There was an extremely poor response on the part of the bone marrow. The leucocytes numbered 16,800 per cubic millimetre, 85% being polymorphonuclear cells, 11% lymphocytes and 4% monocytes. The blood calcium content varied between 9·3 and 11·0 milligrammes per 100 cubic centimetres of serum, and the blood phosphorus content was 4·7 milligrammes. The urine was acid and free from albumin and sugar.

Dr. Macintosh remarked that the child's progress had been slow; periods of some improvement had occurred, and other periods in which progress was far from satisfactory. The response to treatment had been slow. The child's weight was at the time of the meeting 6·0 kilograms (thirteen and a half pounds) and had been 6·5 kilograms (fourteen and a half pounds) since his admission to hospital. All antiscorbutic measures had failed to cause rapid improvement. These included fruit and vegetable juices, potato cream *et cetera*, also "Radiostoleum" and "Redoxon" given intravenously, intramuscularly and by the mouth. The condition three days before the meeting became worse, and a blood transfusion was given with beneficial results.

Juvenile Familial Cerebro-Macular Degeneration: Spielmeyer-Vogt's Disease.

DR. D. G. R. VICKERY showed two brothers, aged eleven and nine years, whose father and mother were first cousins. There had been one other child, which was stillborn. There was no past history of the disease in any other members of either family; they were not Jewish. The mother stated that since the age of six years both the children's eyesight had become defective. At nine years of age convulsions occurred, and there had been great mental deterioration since. The children appeared perfectly normal up to the age of six years. Both children walked at twelve months and talked at eighteen months of age.

On examination both children appeared to be normally grown for their age, but walked with a characteristic stoop of their shoulders, peering as if to distinguish objects in the dark. The expression and attitude were somewhat that of a Parkinsonian syndrome. Their vision was very much impaired, especially the central vision, which appeared to be absent. Definite mental changes had occurred in both children. Attention, memory and judgement were all impaired. There was lack of emotional control, and the speech was stammering, with repetition of words and syllables. The changes were more pronounced in the older boy. Convulsions commenced in the older boy eighteen months prior to the time of the meeting, and every three months he had three to six convulsions per day. The pupils were equal and reacted to light and accommodation. The knee jerks were difficult to elicit. The plantar reflexes were flexor in type, and the abdominal and cremasteric reflexes were sluggish. In the elder boy a positive Kernig sign was present. There were some unsteadiness in walking, and early spasticity of the limbs was noticed. There was no response to the Wassermann test.

Examination of the fundi of both children by Dr. Stanton-Cook revealed right-sided and left-sided optic atrophy, macular degeneration and pepper spots over the whole of the retina.

Dr. Vickery remarked that the condition of the two children was typical of cases described as the juvenile form of cerebro-macular degeneration. The disease was familial, and as a rule more than one child in a family were affected. It was not confined to the Jewish race. A history of similar symptoms in a previous generation might be elicited. The parents were often related, and in this case they were first cousins. The disease appeared to be dependent upon a recessive hereditary factor. It first made its appearance at between four and ten years of age, the first symptom complained of being loss of vision. Several years later convulsions and then progressive mental

degeneration occurred. Later a spastic type of paralysis and a Parkinsonian type of syndrome developed. The patients became bed-ridden, and intercurrent infections completed the story.

Dr. Vickery further said that the pathological findings in this condition were similar to those found in Tay-Sach's disease. The cerebrum usually showed no malformation or gross changes. Histological examination revealed universal changes in the ganglion cells throughout the entire central nervous system. The cells were swollen and their cytoplasm presented a hyaline appearance. The Nissl bodies were almost absent or confined to very fine granules. The swelling was due to deposits of large and small lipoid droplets in the cytoplasm. More or less extensive demyelination of the dendrites occurred later; this seemed to be secondary to the destruction of the cells. There was no evidence of any inflammatory process nor any significant change in the blood vessels. The process in the retinæ was identical with that in the brain. The ganglion cells, which were most numerous in the region of the maculæ, became swollen and finally necrotic, causing the retina to assume a dirty-white appearance. The optic nerves showed secondary degeneration. The condition should be differentiated from congenital syphilis with chorioidoretinitis and from pigmentary degeneration of the retina. The last condition did not cause mental degeneration or convulsions. Dr. Vickery pointed out that the two cases under discussion were following the typical course of the disease; the onset was at approximately six years of age, with failure of vision, then convulsions and mental degeneration, and finally a generalized spastic paralysis with amentia.

Punctate Epiphyseal Dysplasia.

Dr. Vickery next showed a female infant, aged three months, who had been born with deformity and stiffness of most of her joints. She was a full-time baby and the birth was normal. During the first few days of life the child took a number of "blue turns" and oxygen had to be administered. She was unable to suck during the first few weeks of life, but at the time of the meeting was fully breast fed. There were two other children, aged eight and six years, who were healthy.

One other child, born three years previously, with a similar condition to this child, died at nine months of age from intercurrent acute pyelitis. Various splinting apparatus had been used in this case to endeavour to straighten the deformed, stiff joints, but with very little success.

On examination the child appeared abnormal and stunted in growth. Her head was small, with frontal and parietal bossing, and her eyes wandered around aimlessly. She had double congenital cataract. Most of the larger joints were swollen and movement was much limited. There appeared to be no pain or tenderness, except on forced movement of the joints. The proximal segments of both limbs (upper and lower) were short, as in achrondroplasia. The X ray film was typical of the condition, revealing shortened proximal segments of the limbs and pronounced stippling of the epiphyseal ends of all the long bones, with extensive disorganization of the joint surfaces. There was no response to the Wassermann test.

Dr. Vickery remarked that a very limited number of these cases had been reported in the literature. Pritchard was credited with having described the first case, and the disease had been referred to as "Pritchard's disease", "atypical achondroplasia" and "stippled epiphyses with congenital cataract". Dr. Vickery was of the opinion that this was the first occasion on which the disease had appeared in two members of the same family. It seemed that the condition was a congenital disorder of the growth of cartilage, and the defect had to be sought for in the pituitary gland or in some other hormonal influence. Both these patients appeared to be mentally deficient. In the first case the brain failed to develop normally during the nine months of life, and the child's skull measurements were very much below normal, the circumference being 37·5 centimetres (fifteen inches) at nine months of age, although the head was "bossed", in contradistinction to the true microcephalic head, which was peculiarly devoid of bossing and "bumps". Both children had congenital cataract. Dr. Vickery thought that it would be interesting to know the histological structure of these cataracts.

Multiple Congenital Defects.

DR. E. H. M. STEPHEN showed a boy, aged fourteen years, who was the third of four children. He had previously had whooping cough and a secondary anæmia. He was born with cataracts and heart disease. His speech was imperfect; his vision appeared normal, although alternating strabismus with nystagmoid movements in all directions was present. The reflexes were exaggerated, and the child walked with a spastic gait. Examination of the circulatory system disclosed bulging of the precordium. A loud, harsh systolic murmur was heard in all areas; it was conducted into the axilla and neck, and was heard all over the chest. A systolic thrill was audible in all areas, and cardiac dulness was increased.

Gastro-Enteritis.

Dr. Stephen's next patient was a female infant, aged fifteen months, who had been ill for two days. No vomiting had occurred, but diarrhœa was present, the child having twelve motions during the night. The stools were green and contained blood and mucus. The child was feverish.

On examination the child appeared ill and slightly sunken-eyed. Slight dehydration of the abdomen was noted, but the other systems were normal.

Dermatomyositis.

Dr. Stephen then showed a boy, aged nine years, the oldest of four children, the other three being healthy. Two years previously he had complained of pain in the back and calves; it then developed in the arms, accompanied by swelling of the face, legs and arms. He had been unable to bear pressure on the affected parts. The hands, face and legs had become a purple colour. On examination the patient had been found to be a well-nourished child, with a bluish coloration around the eyes and a little œdema of the face. The muscles were very tender on pressure, and the child was able with difficulty to sit up, but was unable to walk. His condition had gradually improved; the tenderness and heliotrope colour had disappeared and the child had become able to walk.

The present illness consisted of pain and swelling in the forearms of three months' duration; swelling of the chest had occurred, and at the time of the meeting the legs were affected. A heliotrope tint was seen around the eyes and on the legs, and the abdominal muscles were tender.

Lymphangiectasis.

Dr. Stephen's next patient was a girl, aged six years, who had had nævi on both legs since birth. She had suffered from recurrent attacks of pyrexia, from pain in the foot, from vomiting and from swelling of the legs in the region of the nævi. She had previously had radium treatment to the sole of the foot.

Brownish-purple areas level with the skin were visible, extending over the back and front of both legs, and onto the abdomen in the region of the *symphysis pubis* and over the lumbo-sacral area. Slight swelling of the lower part of the left leg was also seen. No œdema or tenderness was present. The superficial veins were very much dilated, but the other systems were clear.

Cœliac Disease.

Dr. Stephen next showed a female patient, aged seven years, who had been admitted to hospital on February 22, 1938, with long-standing cœliac disease. For the last few weeks prior to admission to hospital she had been tired and listless, had suffered from anorexia and was losing weight.

On examination the child was seen to be small for her age. She was pale and listless, and her limbs were thin and poorly developed. The tone of the abdominal muscles was poor and the abdomen was protuberant. It had a

generalized "doughy" feel. The child's weight was 14·0 kilograms (thirty-two pounds).

Examination of the fæces on February 25, 1938, revealed that the total fat was 12% of the dried fæces, and of this 25% was unsplit fat. Examination of the blood on February 28, 1938, revealed 3,890,000 erythrocytes per cubic millimetre; the colour index was 0·92. No abnormality was detected in the leucocytes. X ray examination of the chest revealed no abnormality.

The child was given a special diet, the total calories being 1,602; she received 280 grammes of carbohydrate, 47 grammes of protein and 33 grammes of fat. In addition, she was given dilute hydrochloric acid three times a day, an intramuscular injection of two cubic centimetres of "Campolon" weekly, nine drops of "Halivol" daily, and 0·016 gramme (one-quarter of a grain) of thyreoid extract twice a day.

Rheumatoid Arthritis.

Dr. Stephen finally showed a girl, aged three years, who had been admitted to hospital on January 14, 1938. Following congestion of the lungs, from which the child had suffered ten weeks previously, swelling of the hands and wrists and pain in the neck had developed.

On examination both wrists were pale, tender and swollen; movements were painful and impaired. The interphalangeal joints were slightly swollen, but movements were not restricted. Movements of the cervical vertebræ were voluntarily restricted and passive movement caused pain. No abnormality was detected in the heart. The tonsils were enlarged and slightly reddened.

A blood count was done on January 24, 1938, and revealed that the erythrocytes numbered 3,250,000 and the leucocytes 11,600 per cubic millimetre. The colour index was 0·68. Of the leucocytes, 74% were polymorphonuclear cells, 18% were lymphocytes, 7% were monocytes and 1% were eosinophile cells.

The urine was found to contain albumin and *Bacillus coli communis*.

The patient was given a course of intramuscular injections of "Solganal"; she also received dilute hydrochloric acid three times a day, liver extract, and on March 7, 1938, a transfusion of 150 cubic centimetres (five ounces) of citrated blood.

Up to the time of the meeting the patient's temperature had been periodically irregular, rising to 38·35° C. (101° F.). The joints were still swollen and movement was impaired, though to a much less degree than formerly; but the joints were not tender. The patient's general condition was very much improved.

Renal Calculus.

Dr. P. L. Hipsley showed a boy, aged six years, who had had recurrent hæmaturia for nine months. Great loss of weight had occurred during the five months prior to the child's admission to hospital on March 10, 1938.

On examination the boy was seen to be pale; no œdema was present. No tenderness or rigidity was detected over the renal areas; neither kidney was palpable. No abnormality was detected in the cardio-vascular, respiratory or alimentary system.

On March 14, 1938, a plain X ray examination revealed a large stone in the pelvis of the left kidney and a dendritic second stone in the lower and middle calyces. On April 9, 1938, examination of a 24-hour specimen of urine revealed no tubercle bacilli. On March 14, 1938, a blood count was done; erythrocytes numbered 4,700,000 and leucocytes 9,000 per cubic millimetre. On March 13, 1938, the urine contained much albumin but no pus; organisms (*Bacillus coli communis* and enterococci) were present in large numbers. On March 23, 1938, the blood urea content was 20 milligrammes per 100 cubic centimetres. On March 26, 1938, an excretion pyelogram revealed a large "staghorn" calculus on the left side, with hydronephrosis; pyelonephritis was present. The bladder and the right kidney were clear.

At operation on March 29, 1938, through a curved renal incision, the pelvis of the kidney was incised and the stones were removed. A tube was inserted through the kidney substance into the pelvis, the pelvis was sutured and the wound was closed, tube drainage being provided. From March 29, 1938, to April 9, 1938, the patient passed bright blood in the urine. The stones were found to be composed mainly of phosphates, with some urates and organic material. On April 3, 1938, the tube was taken out of the kidney. On April 8, 1938, a transfusion of 240 cubic centimetres (eight ounces) of citrated blood was given intravenously. On April 9, 1938, the blood calcium content was 11·2 milligrammes per 100 cubic centimetres. On April 12, 1938, urine was draining from the wound and the child was passing urine containing much pus. A blood count made at this time revealed 3,460,000 erythrocytes and 10,950 leucocytes per cubic millimetre. The hæmoglobin value was 68%.

Malrotation of Bowel.

Dr. Hipsley next showed a boy, aged six and a half years, who had not been well for the previous three years. He had complained at intervals of pains in the abdomen accompanied by vomiting.

On examination he was seen to be pale; no masses, tenderness or rigidity were detected in the abdomen. The liver was barely palpable; the spleen and kidneys were not palpable. No abnormality was detected in the cardio-vascular or respiratory system.

At operation on March 29, 1938, the caecum and loops of large bowel were freed from the left hypochondriac region and replaced in the abdomen in their normal relationship to the small bowel. The caecum was fixed in the right iliac fossa. On April 2, 1938, the bowels were well open, and on April 12, 1938, the sutures were out and the wound was clean.

Multiple Chondromata.

Dr. H. G. Humphries showed a boy who had a history of swellings on the bones since infancy; the swellings had been gradually increasing in size. One large one was excised from the anterior part of the seventh rib on the right side at the age of two years. There had been no sign of a recurrence. One tumour behind the right knee was growing so big that it caused gross swelling of the calf.

On examination multiple tumours were found. On the right tibia a hard, fixed, bony tumour was present over the upper third on the posterior aspect. The diameter of the calf 10 centimetres (four inches) below the joint line was 30·6 centimetres (twelve and a quarter inches); on the left side it was 26 centimetres (ten and a half inches). Small tumours were present on the medial aspect of the upper third and on the anterior part of the distal end of the tibia. One tumour was present on each side of the distal end of the right femur and one posteriorly. There were two tumours on the anterior surface of the right tibia and one on the medial aspect of the distal end of the right femur. Tumours also occurred on the eleventh rib on the left side and on the upper third of the left humerus, on the outer part of the left clavicle.

X ray examination of the right tibia revealed multiple exostoses present at the ends of the long bones, and osteochondromata in some of these situations as well. Pronounced diaphyseal aclasia was also observed.

At operation on April 8, 1938, a large lobulated tumour of the posterior aspect of the upper third of the right tibia was removed.

Renal Rickets.

Dr. C. L. S. Macintosh showed a female, aged twenty-two years. Her mother had suffered from "kidney trouble" just prior to the patient's birth; a sister suffered from Addison's disease, and another sister from pulmonary tuberculosis.

The patient was the third child of twelve. She suffered from "wasting disease and gastro-enteritis" as an infant, from chicken-pox at the age of two years, and from mumps when seven years old. When she was five years old her mother noticed the appearance of "dark spots" over her body generally.

She had first come under notice in 1930. Up till two weeks before that time her general health appeared to have been satisfactory. She was suffering from more or less constant headaches, was becoming pale and sallow, losing weight and becoming weak. She became thirsty and was passing an increased amount of urine.

On her admission to hospital on February 4, 1930, she was an undersized, knock-kneed, thin and anæmic-looking child. Pigmented areas or spots of varying size were seen about the body generally, and more especially about the face and head. On the trunk, and especially on the anterior abdominal wall, were small "white areas", much paler than normal skin. The lower limbs appeared to be rotated inwards, giving a marked degree of *genu valgum*, and she walked with an alternate crossing of the knees. The liver and spleen were not enlarged. The sex development was delayed. Her intellect was fair.

The pigmented areas on the skin, examined microscopically, were seen to be due to increased deposits of normal skin pigment. The "white areas" were *nævi anæmici*. There was no response to the von Pirquet and Wassermann tests. The urine at times was faintly clouded with albumin, but usually it was albumin-free; it was never found to contain sugar. The specific gravity was low, usually from 1,002 to 1,010. The amount was from 568 to 1,136 cubic centimetres (20 to 40 ounces) *per diem*. Nocturnal frequency of micturition occurred on occasions.

Blood examination revealed 4,010,000 erythrocytes per cubic millimetre; they were pale and regular in shape and even in size. The hæmoglobin value was 42% and the colour index 0·5. The leucocytes numbered 5,000 per cubic millimetre, the polymorphonuclear cells being 73%, lymphocytes 26%, monocytes 10% and eosinophile cells 1%. Blood platelets were numerous. Dr. Macintosh pointed out that various blood counts done for the patient were very similar to the one quoted. The blood urea concentration varied between 30 and 64 milligrammes per 100 cubic centimetres, and at the time of examination was 34 milligrammes per 100 cubic centimetres. The blood cholesterol content was 145 milligrammes per 100 cubic centimetres. The blood calcium and blood phosphorus content varied from time to time. Dr. Macintosh said that on February 19, 1930, the blood calcium content was 6·6 milligrammes and the blood phosphorus content was 7·9 milligrammes per 100 cubic centimetres of serum; on May 27, 1930, the blood calcium content was 10·0 milligrammes and the blood phosphorus content 12·6 milligrammes per 100 cubic centimetres; on April 23, 1931, the blood calcium content was 9·0 milligrammes and the blood phosphorus content was 8·3 milligrammes per 100 cubic centimetres; on August 11, 1931, the blood calcium content was 11·4 milligrammes and the blood phosphorus content was 1·2 milligrammes per 100 cubic centimetres; this result was checked, and could not be accounted for; on March 5, 1933, the blood calcium content was 9·0 milligrammes and the blood phosphorus content was 3·2 milligrammes per 100 cubic centimetres. At the time of the meeting the phosphorus content was 5·4 milligrammes per 100 cubic centimetres.

The blood pressure was normal.

X ray examination of the long bones revealed changes in the ends of the diaphyses, about the knee and ankle joints, typical of renal rickets. In the radii and ulnæ slight changes were apparent, especially at the lower end of the ulnæ. X ray examination of the skull revealed small irregular areas of osteosclerosis, typical of early Paget's disease.

At the time of these investigations the patient was thirteen years old. Dr. Macintosh said that this was several years younger than those in whom Paget's disease usually made its appearance. Further, in renal rickets changes not unlike those of Paget's disease appeared. Examination of both antra revealed pronounced chronic hyperplasia of the lining membrane, the result of infection. No definite changes were seen in the spine.

The sex development had been delayed, but was apparently normal, menstruation having begun when the girl was sixteen years old. The *genu valgum* had been treated by operative and orthopædic measures, with excellent results. Frequent and severe headaches and back pains had been troublesome since 1930; at times the pains extended into the legs. Dr. Macintosh pointed out that age in this case was of interest, for in most cases the patient died in the second decade from uræmic states.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Atkins, Walter Thomas Garner, M.B., B.S., 1937 (Univ. Sydney), Kildare Street, Blacktown.

Christie, Joseph, M.B., B.S., 1935 (Univ. Sydney), 80, Queen Street, Auburn.

Jakins, Leonore Edith, M.B., B.S., 1936 (Univ. Sydney), Bateman's Bay.

Johnson, Bristow, M.B., 1935 (Univ. Sydney), 261, Elizabeth Street, Sydney.

McEntire, Ronald Gordon John, M.B., B.Ch., 1912 (Univ. Dublin), B.A.O., 225, Macquarie Street, Sydney.

O'Reilly, Vincent John, M.B., B.S., 1936 (Univ. Sydney), Saint Margaret's Hospital, Bourke Street, Sydney.

Rawle, Ronald Maxwell, M.B., B.S., 1930 (Univ. Sydney), F.R.C.S. (England), 1935, 143, Macquarie Street, Sydney.

Correspondence.

THE COPPER SULPHATE TREATMENT OF TROPICAL ULCER AND NEW GUINEA MOUTH DISEASE.

SIR: I have read with great interest Dr. Gunther's paper on tropical ulcers in a recent issue of your journal. I am keenly interested in this most important subject. My experience of nine years spreads over three parts of Melanesia, the worst ulcers occurring in New Britain, Mandated Territory of New Guinea. It was not infrequent for a child to die of an ulcer of only one week's duration. In fact, ulcers, with their complications, accounted for the most deaths in my hospital there. In the western and central Solomons ulcers are of a less serious variety, though bad ones occur there also.

In treatment of these serious cases, applications are insufficient. Curetting or excision is often necessary, followed by the application of a strong solution where the eradication of sepsis is in doubt, or by skin graft where asepsis is sure. In the former, seed grafts are put onto the clean granulations subsequently. I described the method in *The Lancet* (1932).[a]

Dr. Gunther strongly recommends copper sulphate (1/150) and considers it to be specific. I have used this drug for a long time now, but by a rather different method, that is, using a strong solution so that the caustic effect of the copper sulphate comes into action.

A comparison of the two methods was made.

Ramonga, male, twenty years, had an active progressive ulcer, and was admitted to hospital. Fusiform bacilli, spirochætes and filamentous forms were numerous on a smear. From past experience I considered that two applications of the solution I use would clean it up, and adhesive plaster be applied about the fourth day. Dr. Gunther's instructions were followed for four days, when there were still numerous organisms present. This was unsatisfactory. Next morning and evening my stronger solution (see later) was applied, and followed by the usual acriflavine foment. Next day there were no organisms on a smear. The following day, scarlet red and plaster were applied. Healing rapidly took place.

This experience, one of very many, confirms me in recommending the following method of treatment.

Copper sulphate, one ounce.
Glycerine, two ounces.
Acid carbolic, one drachm to the ounce of resulting solution.

Crush the copper, put it and the glycerine into an enamel bowl and heat. Stir, allow to cool, and pour off the solution, leaving any undissolved crystals in the bowl. Add one drachm of carbolic to each ounce of solution. The carbolic is an anæsthetic and diminishes the sting of the copper, and the glycerine enables a strong and stable solution to be obtained. This is not the case with water.

Method of Use.—Gently clean the ulcer with wool swabs. Place the limb horizontally, and apply the solution on a piece of wool rolled to the size of a pea. Continue the application until the surface feels hard and granular, that is for, say, two to three minutes. Only good can come of prolonging the application, and should any run over onto healthy skin, though best avoided, it is innocuous. Then put on a small dressing of acriflavine on gauze and wool (1/1,000) and cover it with a hot foment and protective. This is soothing, cleansing, and reduces the swelling. The same evening, the ulcer may or may not be "messy" as the result of the morning's application. Repeat the application then or not until the morning, according to the severity of the ulcer. Put on the acriflavine foment. On the next morning, the ulcer is usually clean and aseptic. If still a small area of dark colour, repeat application. Most ulcers are clean after applications on two consecutive mornings. On the fourth morning, scarlet red ointment and adhesive plaster can be applied.

The more serious ulcers need curetting first before the application. A tourniquet is necessary also.

Other details may be found in my small book, "Diseases Commonly Met With in Melanesia" (Melanesian Mission, Sydney).[2]

Yours, etc.,

CLIFFORD JAMES, M.B., Ch.B. (New Zealand), D.T.M. & H. (London), F.R.C.S. (Edinburgh).

The Hospital of the Epiphany,
Fauabu,
North Mala,
British Solomon Islands.
May 25, 1938.

References.

[1] C. James: "Tropical Ulcers: Treatment by Excision and Skin Graft", *The Lancet*, Volume IV, November 19, 1932, page 1095.
[2] C. James: "Diseases Commonly Met With in Melanesia", 1937, page 19.

PHYSICAL THERAPY IN OTO-LARYNGOLOGY.

SIR: The increasing recognition of the importance of sinusitis must surely lead to an improvement in its treatment. Dr. Gutteridge's paper on physiotherapy and sinusitis pleased me more than any other for over twenty years. I am not going to quarrel with him over his sneer about long-wave diathermy; the important point is that he recommends some kind of diathermy. If he would only proceed further and do antrostomies by electrocoagulation I am sure he would use no other means in 90% of his cases to overcome sinusitis in old and young.

Surgical antrostomy is generally useless, and especially so in children. It is often tedious and difficult to perform properly, whereas with electrocoagulation it is a question of seconds and is as easy as lavage; more important, it seems to me to last indefinitely when thoroughly carried out. Insert your needle into the antrum and twist it up and down, forwards and backwards until you have electrocoagulated a wide area; and as you withdraw your needle be careful to electrocoagulate the outer wall of the inferior turbinate. In adults often a smart tap is necessary to push the needle through the wall at the proper place, as it is most important to destroy the lower part of the antro-nasal wall and so obtain a permanent opening low down and as near as possible to the angle between the facial and nasal walls of the antrum.

Some years ago I reported a cure of nasal polypi protruding through the anterior nares in a man of forty-three, by diathermy only. I have only obtained a cure in one other case, a boy of fourteen, but most of my cases have been previously operated upon, and for some reason, probably infiltration, one cannot cure them by means of physiotherapy.

Except in acute cases, I consider lavage not only useless, but harmful, and I still maintain, after eighteen years' experience, that the large majority, at least 75%, of enlarged tonsils and adenoids in children can be cured without any other treatment than physiotherapy, antrostomy and nasal hygiene.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
May 21, 1938.

NATIONAL HEALTH INSURANCE.

SIR: I have just read the leading article in *The Sydney Morning Herald*, and as it is one of the most authoritative papers printed, I think that the hope of any reasonable terms is almost negligible. The facts are that in Australia there are too many doctors in practice (5,200 to 7,000,000), the income limit is far too high (£350), owing to the hospital contribution scheme with its implied contract practice (which should have been rigorously suppressed by the Council) the people have been educated to expect all treatment on a small contributory payment, the overhead and costs of living are so high that a most experienced and successful practitioner gave to me as his considered opinion that a practitioner had to take £1,200 before he saved anything. Since then taxation has increased out of all proportion.

Deducing from the above data we arrive at the sad facts: at the present scale of payment there are not enough patients to go round, that the extra items will be largely unpaid for (in an industrial practice the senior partner told me that he was then confining women for whose confinement he had never been paid, and that he hoped soon to have a grandchild on the same terms), and with the increase of taxation which everyone must expect doctors as a species face extinction.

Now it is common knowledge that with the large defence commitments, the expense of dealing with a large influx of immigrants, the large amount of interest that has to be paid on the national debt, the expensive public works that must be undertaken to develop this country, and the pension burden that is looming high over us all, we cannot expect a scale of payments that will give us anything like the return of pre-depression days.

In view of the above I think that we should explore the conditions of nationalization of medicine, as they may be much better than we have been led to believe.

During this few months the council of the Association has not been seen in a good light. To have entered into an agreement not to reveal the terms of its agreement to those most affected until they were quoted in Parliament (particularly as they surely must have realized how unacceptable to the members they would be) was surely a most reprehensible act. In addition the way they have condoned the contributory hospitals schemes and allowed the public to be educated to expect treatment in the same manner as one expects to ride on a ferry-boat with a season ticket has debased the practice of medicine, and it is now as much a business as selling groceries. Soon I expect we will all be advertising.

In conclusion, there is a letter in *The Sydney Morning Herald* which is the most able and dignified presentation

of the position that I have read, and I enclose same with suggestion that you should print it. Under the nationalization of medicine or under the national insurance scheme we will become mere clerks; but as there is evidently no choice in the matter I prefer that we should at least be under the ægis and protection of a Government department.

Yours, etc.,

THOMAS J. RITCHIE, M.B., B.S.

Burren Junction,
New South Wales,
May 31, 1938.

SIR: In a statement apparently authorized for publication by the Federal Council, which appeared in *The West Australian* of May 23, our representatives placed on record the features which would be desirable in an adequate scheme of medical service. It is, however, to be regretted that their criticism of the Government scheme, which is admittedly the result of a compromise between the views of the Federal Council and the Government, should include a serious misstatement. For medical benefit, under this bill, not only does not ignore preventive measures, but it was specifically designed to provide scope for such. Moreover, it does provide vastly increased opportunities for the early diagnosis of disease.

It was hoped that the abolition of the doctors' bills would encourage the insured workers to consult their medical advisers at an earlier phase of their illnesses, or even to pay them periodic visits while well, in order that the trained observation of their doctors might detect signs of incipient disease before any symptom had been observed by the "patient", and thus many periods of incapacity might be avoided. The experience of panel doctors in England, however, has been that the removal of the fear of the bill has not sufficed to bring about the wished-for change, as the convention not to go to the doctor till one is "really ill" clearly goes deeper than financial considerations.

It is certainly time that doctors, who are fully alive to the importance of earlier opportunities for the detection of disease, should take steps to become associated in the minds of their public more with the building of health than the treatment of disease. For this reason alone we should welcome the coming of contract practice on a well-organized scale and set ourselves to exploit its provision under medical benefit of the facilities for the prophylactic practice of our craft. And since our remuneration under the contract is unaffected by the continued health of those on our list, it is to our pecuniary profit to do everything in our power to lessen the incidence of sickness among insured workers on those lists.

Yours, etc.,

S. BOYD.

Gnowangerup,
Western Australia,
May 26, 1938.

SIR: The national insurance is founded on false bases. The Federal Government conveniently assumes that payment under the lodge agreement is profitable to doctors. It was never expected to be profitable. The average country practitioner has forty to two hundred lodge patients. It is only when the numbers reach five hundred or more that it becomes payable. The lodge agreement resulted from an endeavour by working people to pay what they could, and has been honourably kept.

Again, the Federal Government's idea of a general practitioner service is sixty years behind the times. The old general practitioner disappeared long ago. The present-day general practitioner is intensively educated, expensively equipped, and in the country does all major surgical and gynæcological work and many special operations—giving a very high degree of service to the community. His electrical and X ray equipment often costs from £400 to £1,200. The Federal Government should wake up and recognize that while it is sixty years behind the times the doctors have steadily progressed.

We are offered 11s. a year, or 2¼d. a week; one telephone message to hospital would about absorb this. And for this we are expected to work twenty-four hours a day, 365 days a year. Most country associations have already rejected the terms, and usually unanimously.

The Federal Government threatens to supplant us with Government pets or importations. The country practitioners of New South Wales will stand firm for 20s.; those beyond the 200-mile limit should not accept a penny less.

To frame this act on an English basis is ridiculous; cars, petrol, equipment in Australia cost twice as much.

The financial clauses have been made possible by exploiting the doctors, whose just claims have been brutally thrust aside.

It seems, however, that the attempt will fail. The general practitioners, and especially country general practitioners, must strongly organize and, if the insurance act later on extends, insist that it must insure the doctor a day not exceeding twelve hours.

Yours, etc.,

E. R. ROSEBY, M.B., Ch.M.

Boggabri,
May 30, 1938.

SIR: When Sir Walter Kinnear was invited by the Federal Government to report on the possibility of establishing a national health insurance scheme in Australia it was confidently expected that the scheme would follow the British system in as many points as possible. So far nothing has eventuated to alter that opinion; both his report to the Government and the proposed bill for the act follow as religiously as possible the British scheme, with little or no modification for our different conditions. In view of these facts it is evident that the rules and regulations governing the panel doctors in England will need close scrutiny; for it is certain that unless a strong stand is taken by the profession in Australia the regulations will be modelled as closely as possible upon the British ones.

From the point of view of the general public, the British scheme is, in theory, ideal. Excluding confinements, there is virtually no service that the public cannot expect to receive from their panel practitioner, on whose list their names may be placed, without extra charge. In England it is a ruling that once a service has been rendered it will be presumed to be treatment within the scope of the insurance contract unless the practitioner can prove otherwise.

When away from home, either travelling or on holidays, any insured person is entitled to demand attention from any panel practitioner without fear of being charged a fee. In addition, when any emergency arises, the public themselves being the sole judges of what constitutes an emergency, and their own doctor not being readily available, the insured person is entitled to the services of any panel practitioner without charge.

If any person is dissatisfied with the attention received and is desirous of making a complaint, he has an approved society willing and capable of preparing and pressing his case, and when necessary, employing legal aid on his behalf.

From the medical point of view the position is not so satisfying. The remuneration is poor and the work is all-embracing. Small though the English capitation fee is, it does not follow that any doctor receives the 9s. per person per year for every name on his list. The number of persons on insurance is actuarially calculated in advance each year. This number is multiplied by 9s. and this is the total amount of money available for distribution to the profession for medical benefits for the current year. This is divided among the various insurance committees for distribution to the medical profession, and apportioned among them according to the known number of persons on each doctor's list. Certain sums are deducted from this money prior to being apportioned to the panel practitioner, such as anæsthetic fees and panel committee expenses, and each doctor's cheque is adjusted, either being

increased or diminished, for emergency services and attendance upon temporary residents. It will thus be seen that any additional payment made to a doctor for extra work performed is made at the expense of a fellow practitioner, and not paid for by the insurance authorities.

No allowances are made for people moving in or out of insurance during any quarter or transferring from one doctor's list to another during a quarter. Unless a name is on a doctor's list within forty-eight hours of the commencement of a quarter, he receives no financial credit for attention given during that quarter. This, of course, makes calculation for payments to doctors much easier for a central bureau, but ignores the claims of the profession to be paid their just dues for all services that they are compelled to render.

Prescriptions must be written in duplicate and no reference is permitted to a previously issued prescription. If a doctor orders an appliance on an official form, this is supplied at the insurance trust's expense, but if it is later decided that the patient should have paid for the appliance, the trust is recouped at the doctor's expense. With regard to sick pay, there are seven different certificates that may have to be used. If an anæsthetic is given to an insured person, a report has to be made, as is the case if a claim is made for payment for special services outside the scope of insurance practice. In both these cases full details of the patient's clinical condition and the reason the services were rendered have to be supplied. Clinical records have to be kept and every visit or attendance to an insured person recorded.

Tuberculous patients are the subject of special departmental investigations, and there are three forms that may have to be filled in in this respect.

The position of a panel practitioner with regard to the investigation of complaints is particularly trying. Any insured person or his representative, an approved society, or an insurance committee, may make charges against a doctor, and these are investigated by a subcommittee. This subcommittee reports to the insurance committee, which may, if it thinks fit, advise the minister to withhold portion of a doctor's quarterly cheque. The doctor has the right to appeal to the minister against an adverse decision and on such appeals both sides may have legal assistance. The penalties to which a doctor may be subjected range from a fine of £5 to removal from the panel.

These, briefly, are the salient features of the British scheme, and it needs little imagination to see that the dice are heavily loaded against the medical profession and strongly in favour of the insured person and the departmental authorities.

Yours, etc.,

LESLIE ABRAMOVICH.

334, Canterbury Road,
Canterbury,
New South Wales.
June 15, 1938.

SIR: Is it a fact that national health insurance will reduce the income of numbers of doctors to one-half and that some are going to be forced out of practice altogether? Let us see if this is an exaggeration.

The Government is setting out to buy medical service for several millions of the lower paid classes and naturally wants it at the lowest possible price. After taking a few furtive soundings and a little kite-flying it offers what it claims is the average of the price of contract practice, namely twopence halfpenny per week per insured person. By including dependants in the scheme four million people become panel patients, and of these, two and a half million are at present private patients, receiving private patient courtesies and paying private patient fees. They are going to save about a million pounds in the aggregate and their doctors are going to lose it.

Imagine the case of a doctor whose patients are all private, but who will come under national insurance, paying him 2s. 2d. per call (and that 2s. 2d. is simply the Government's capitation fee of 11s. *per annum* divided by the Government's estimate of five services per year). If his private practice averages no more than 6s. 6d. per call, his income is reduced to one-third! And his expenses are not reduced one iota!

The two-guinea specialists, the doctors on fixed salaries, and those depending mainly on lodge work, will escape at first, and the private-patient doctors will have to take the brunt of the attack. Numbers of them will be driven out. Where to? Some will specialize, some will move to industrial districts where panel lists are bigger, some will retire, and so on.

Now this reduction of the national medical income by about one million pounds threatens the professional existence of those doctors whose practice includes an appreciable proportion of private patients (liable for national insurance), and the onus is upon them to defend themselves. What can they do? Here there is precedent aplenty. Many unions have learnt the technique of resisting attacks on their wages and working conditions, through years of experience. They first appoint a war council, consisting entirely of known fighters, and only the best are considered good enough. Half-hearted fighters are superseded. They choose only men who have common cause with them. Men who will suffer with them, men whom we will find among the middle-class private-patient general practitioners. If we want our adversaries' respect we must have the best generals to lead our attack. Next we will need ammunition. What capitation fee would represent: (*a*) general practice, (*b*) private practice? On what figures can we estimate "fair and reasonable" capitation fee? Here we need a good actuary. And to fire this ammunition we need the best advocate that money can buy. Or is it all too late?

Yours, etc.,

"G.P."

June 7, 1938.

MIGRAINE FROM THE ALLERGIC VIEWPOINT.

SIR: Dr. Clive Sippe's paper concerning the allergic basis of migraine (published in your issue of May 21) should suffice to convince any who previously had doubts on the subject. My experience in the past few years has led me to the same general conclusions, and the rapid change in patients whose food sensitiveness has been revealed to them is often most striking. Providing suitable extracts are employed by the intradermal method, skin testing gives very satisfactory results and is not nearly so irksome to patients as test dieting; but, as Dr. Sippe says, the results must be considered in conjunction with other evidence.

The relative frequency of reactions with some of the test substances in Dr. Sippe's series is a matter which invites discussion. It is difficult, for instance, to reconcile the 96% positive result with banana with the usual experience of others dealing with food allergy. Wheat shows a much higher response in my series and those of practically all allergists who have published figures, namely, more like 45%–50%, and the important thing is that changing over to rye flour results as a rule in the relief of symptoms. Egg white, on the contrary, does not react so frequently in my experience, but only in about 50% of cases.

In addition to chocolate, which is mentioned, tomato, rice and lettuce are important factors in food allergy, including migraine. The whole subject of food allergy, including gastro-intestinal symptoms, angio-neurotic œdema (not every case), hives in children, and nocturnal enuresis, has not received the attention in this country which its importance warrants, and therefore Dr. Sippe is to be congratulated on the work he has published.

Yours, etc.,

D. L. BARLOW.

Shell House,
170, North Terrace,
Adelaide.
June 3, 1938.

Obituary.

LESLIE WYLIE NORMAN GIBSON.

LESLIE WYLIE NORMAN GIBSON was the son of William Gibson, who had founded the Scotia wool-scouring works near Brisbane some years before his son's birth on March 3, 1899.

Of the hard-working, upright and capable Lowland stock, which has produced so many great physicians and surgeons, Leslie Gibson carried his inheritance to the Brisbane Grammar School and the University of Sydney. He finished his medical course in 1925 and returned to Brisbane the next year. He held resident appointments at the Brisbane Hospital and Children's Hospital till 1929.

He developed an interest in orthopædic surgery, and resolved to devote himself to it. So at the end of his resident term he made his way to Liverpool—the Mecca of the orthopædic surgeon. Here Sir Robert Jones was still the presiding genius, and through him the influence of Hugh Owen Thomas fell upon Gibson. Like everyone else, he fell completely under the charm of "Robert", whose opinions he always quoted with almost religious reverence. With Sir Robert was his lieutenant and successor McMurray, whose work profoundly influenced Gibson. Another member of this brilliant school was James Eastwood, whose sister Gibson later married. Gibson worked hard at Liverpool and gained the degree of Master of Surgery in orthopædics in 1931. He had impressed his teachers, and both then and subsequently was urged to remain in England. But in 1931, in his absence, he was appointed honorary orthopædic surgeon to the Hospital for Sick Children, Brisbane. He took up duty there in the last months of the year.

His appointment met with much criticism, and his youth and inexperience were freely used to his disparagement. However, in a sharp epidemic of poliomyelitis that broke out in Queensland and lasted from November, 1931, to March, 1932, he answered his critics. Of the one hundred and thirty children treated at the hospital during the epidemic, Gibson saw every one, usually very shortly after admission, and he worked day and night applying splints and plasters. When the epidemic waned his colleagues realized that the hospital had an orthopædic surgeon of no mean quality—one who needed only experience to make him outstanding. With this came his appointment to the medical board of the Queensland Society for Crippled Children, with care of patients in "Montrose", the society's school.

In 1931 he became the very successful honorary secretary of the Queensland Post-Graduate Committee. He held this position for three years, leaving it only to take up the more onerous and responsible post of honorary secretary to the Queensland Branch of the British Medical Association. Later he became a representative of the Branch on the Queensland Medical Board. He continued as honorary secretary of the Branch till the end of 1937, when he relinquished this very heavy work, but remained a member of the council. Meantime his practice was increasing and he became well known.

Unfortunately he was dogged by chronic ill health and often he was interrupted by pain and discomfort severe enough to make work impossible.

In 1935 he was appointed by the Branch to be its representative on the Royal Commission on Poliomyelitis. It is an open secret that much of that commission's report was his work. In his own words, he was determined to make the report "scientifically impregnable". As the only fully trained orthopædic surgeon on the commission, to him fell the task of the repeated muscle testing and checking, the results of which may be studied at length in the report. The work was dull and exacting; it was carried on for two years and was often done in the face of pain and weakness; for at this time Gibson was suffering almost constantly. This report may in fact be looked on as his monument, and although he did not live to sign it, it is well known how great was the part he played in its production.

The long tables of the results of muscle examination and reexamination give the report its outstanding value. No patients were included that Gibson did not see and examine himself, and hearsay evidence was given but a bare reference.

Eventually the progress of his illness made surgical treatment unavoidable. In spite of his colleague's care and devotion, he died on December 23, 1937, at the age of only thirty-eight years.

In his work Gibson held strongly to simplicity and manipulative methods in the tradition of Hugh Owen Thomas and Robert Jones. He had ample courage when necessary, but seemed more content to correct a faulty position with manipulation and plaster than with scalpel and osteotome. He was especially happy in his handling of children, and he had no more sincere mourners than his small patients at Montrose. Deft hands and unlimited patience, with the solid Liverpool training, equipped him thoroughly to take a leading place in his profession.

He has left his widow and two children, to whom we extend our deepest sympathy.

Medical Prizes.

THE STAWELL MEMORIAL CLINICAL PRIZE.

THE Stawell Memorial Clinical Prize, which consists of a money award of £40, is open for competition.

The purpose of the fund from which the prize is given is to commemorate appropriately the memory of the late president-elect of the 103rd annual meeting of the British Medical Association (Sir Richard Stawell) and his great influence in the clinical teaching of medical students and young graduates. Donations to establish the fund were received from members of the medical profession and others, to which was added a grant by the executive of the 103rd annual meeting.

The following are the conditions governing the first award:

1. The memorial shall take the form of an essay. Competition shall be open to Australian graduates of not more than three years' standing on September 15, 1938.

2. The subject of the essay upon which the prize shall be awarded is "The Clinical Significance of Pathological Changes in the Ocular Fundus".

3. The work submitted must be based on personal observations and experiences collected by the candidate in medical practice. A high order of excellence is required.

4. The trustees reserve the right to withhold the award if no essay of sufficient merit is entered.

5. Essays must be in the hands of the trustees, care of the Medical Secretary, British Medical Association (Victorian Branch), 426, Albert Street, East Melbourne, C.2, not later than September 15, 1938.

6. The prize shall be presented at the opening of the Royal Australasian College of Physicians in Sydney during October, 1938.

7. No study or essay that has been published in the medical Press or elsewhere shall be considered eligible for the prize.

8. If any question arises in reference to the eligibility of the candidate or the admissibility of his or her essay, the decision of the trustees on any such point shall be final.

9. Each essay must be typewritten or printed, must be distinguished by a motto and must be accompanied by a sealed envelope marked with the same motto, in which envelope must be enclosed the candidate's name and address.

10. The trustees reserve the right to publish the essay for which the prize is awarded in THE MEDICAL JOURNAL OF AUSTRALIA.

11. Further inquiries relative to the prize should be addressed to the Medical Secretary, British Medical Association (Victorian Branch), 426, Albert Street, East Melbourne, C.2.

NORMAN GAMBLE FUND AND RESEARCH PRIZE.

THE Council of the Royal Society of Medicine has accepted, as a trust, the sum of one thousand pounds presented by Mr. Norman Gamble for the purpose of founding "The Norman Gamble Fund and Research Prize".

The Norman Gamble Research Prize, of £50, shall be awarded for the best original work in otology completed during the four years previous to the month of October in the year of the award. The competition for this prize shall be open to any British subject, whether lay or medical, who cares to compete, whether or not such person be a holder of or candidate for a grant in aid of research.

Grants in aid of research work in otology may be made to any British subject, lay or medical, who may be recommended by the committee of award to receive such grants, whether or not such person be a holder of or a candidate for the Norman Gamble Prize. Moreover, such grants may be given either to persons who apply for a grant in order to carry out a specific piece of work, or to selected workers chosen by the committee of award to carry out certain specified work of its selection.

Any person to whom a grant in aid of research is made from this fund shall, within such time as may be allowed by the ocmmittee of award, render a typewritten or printed report in the English language on the work that has been carried out under the grant, and the committee of award shall have the right to publish such report.

The committee shall not make any recommendation either for the award of the Norman Gamble Prize or for any grant in research if the papers or work under consideration are not of a sufficiently high standard.

The committee of award will consider applications for the prize and for grants in aid of research work in October, 1938.

Applications for the prize and for grants in aid of research must be in the hands of the Secretary of the Royal Society of Medicine, 1, Wimpole Street, London, W.1, England, not later than September 30, 1938, and must be in accordance with the regulations stated above.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on June 6, 1938.

Dr. H. J. Daly and Dr. W. I. T. Hotten were appointed Post-Graduate Lecturers in Anæsthetics at the Prince Henry Hospital.

The following degrees were conferred:

Bachelor of Medicine (*M.B.*) *and Bachelor of Surgery* (*B.S.*): Robert Andrew Duval, Colin Campbell Greenwell, Bazel Henry Louis Grey, William Chalfont Holley, Ronald James Humphrey, Roderick Lionel Jeffrey, Nicholas Larkins, Milton Raymond Lee, Victor Robertson Meek, Doreen Olive Moore, Leswyn Oswyn Sheridan Poidevin, William Vincent Russell.

Bachelor of Medicine (*M.B.*): William Willis Gunther, James Allan Hill, Don Creswell Howle, Eccles Norman Nash.

The following degrees were conferred *in absentia*:

Bachelor of Medicine (*M.B.*) *and Bachelor of Surgery* (*B.S.*): Maxwell Mansfield Brown, B.Sc., Donald Ashley Billing Carter, Patricia Reeves Davey, B.A.

Bachelor of Medicine (*M.B.*): Sidney Brookes Clipsham, Leaton Elias.

Bachelor of Surgery (*B.S.*): Patrick Francis Murphy.

The British Association of Radiologists.

THESIS FOR FELLOWSHIP.

THE following radiologists practising in Australia and New Zealand have obtained the fellowship of the British Association of Radiologists by thesis: Colin Campbell Anderson, Invercargill; Leslie John Clendinnen, Melbourne; Kenneth Stuart Cross, Melbourne; Charles Francis de Monchaux, Sydney; Eric Wilfred Frecker, Sydney; Francis John Gwynne, Auckland; Robert Augustin Gardner, Sydney; Harold John Ham, Sydney; Colin Ferguson Macdonald, Melbourne; Neil McDougall (deceased), Auckland; Valentine McDowall, Brisbane; Alwyn Tom Hays Nisbet, Sydney; John O'Sullivan, Melbourne; Herbert Roy Sear, Sydney.

We are informed that the fellowship will not be obtainable by thesis after December 31, 1938.

Honours.

BIRTHDAY HONOURS.

HIS MAJESTY THE KING has been pleased to confer the following honours on medical practitioners in Australia.

Dr. Robert Blakeway Wade, of Sydney, has been created a Knight Bachelor.

Dr. Augustus Leo Kenny, of Melbourne, has been created a Commander of the Most Excellent Order of the British Empire.

Dr. Vera Scantlebury, of Melbourne, and Dr. Isabella Younger Ross, of Melbourne, have been created Officers of the Most Excellent Order of the British Empire.

The congratulations of the medical profession are extended to Sir Robert Wade, Dr. Kenny, Dr. Scantlebury and Dr. Ross.

Books Received.

CLIMATE AND ACCLIMATIZATION: SOME NOTES AND OBERVATIONS, by Sir A. Castellani, K.C.M.G., D.Sc., M.D., F.R.C.P.; Second Edition; 1938. London: John Bale, Sons and Curnow Limited. Crown 8vo, pp. 208, with illustrations. Price: 10s. net.

CLINICAL ATLAS OF BLOOD DISEASES, by A. Piney, M.D., M.R.C.P., and S. Wyard, M.D., M.R.C.P.; Fourth Edition; 1938. London: J. and A. Churchill Limited. Large crown 8vo, pp. 143, with 42 illustrations, of which 38 are in colour. Price: 12s. 6d. net.

DISEASES OF INFANCY AND CHILDHOOD, by W. Sheldon, M.D., F.R.C.P., with a foreword by Sir F. Still, K.C.V.O., M.A., M.D., LL.D., F.R.C.P.; Second Edition; 1938. London: J. and A. Churchill Limited. Demy 8vo, pp. 751, with 125 text figures and 13 plates. Price: 21s. net.

MENTAL NURSING IN OBSERVATION WARDS, by I. M. Sclare, L.R.C.P.S.; with a foreword by A. S. M. MacGregor, O.B.E., M.D., D.P.H.; 1938. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 260. Price: 6s. net.

SURGICAL HANDBOOK FOR HOSPITAL ASSISTANTS IN THE TROPICS, by W. K. Connell, M.B., Ch.B., F.R.C.S., D.T.M. and H.; 1938. London: John Bale Medical Publications. Medium 8vo, pp. 455, with illustrations. Price: 12s. 6d. net.

INDIVIDUAL PSYCHOLOGY MEDICAL PAMPHLETS. NUMBER 19: THE CONTRIBUTION OF ALFRED ADLER, by P. Mairet and H. C. Squires, C. Dukes, O. H. Woodcock, W. Langdon-Brown and others; 1938. London: The C. W. Daniel Company Limited. Demy 8vo, pp. 72. Price: 2s. 6d. net.

Diary for the Month.

JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 30.—South Australian Branch, B.M.A.: Branch.
JUNE 30.—New South Wales Branch, B.M.A.: Branch.
JULY 1.—Queensland Branch, B.M.A.: Branch.
JULY 5.—New South Wales Branch, B.M.A.: Council (Quarterly).
JULY 6.—Victorian Branch, B.M.A.: Branch.
JULY 6.—Western Australian Branch, B.M.A.: Council.
JULY 7.—South Australian Branch, B.M.A.: Council.
JULY 8.—Queensland Branch, B.M.A.: Council.
JULY 12.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
JULY 19.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 20.—Western Australian Branch, B.M.A.: Branch.
JULY 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 22.—Queensland Branch, B.M.A.: Council.
JULY 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 27.—Victorian Branch, B.M.A.: Council.
JULY 28.—South Australian Branch, B.M.A.: Branch.
JULY 28.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. J. A. Cahill has been appointed Public Vaccinator at North Fitzroy, Victoria.

* * *

Dr. T. Hamilton has been appointed Deputy Quarantine Officer at Newcastle, New South Wales, in accordance with the provisions of the *Quarantine Act*, 1908-1924.

* * *

Dr. M. J. Frizell, Dr. M. Rippin and Dr. B. G. Cooley have been appointed Medical Officers in the Medical Branch of the Department of Public Instruction of New South Wales.

* * *

Dr. H. K. Fry has been appointed an Official Visitor to the Parkside Mental Hospital, Adelaide, South Australia, and a member of the Advisory Committee appointed under Section 17 of the *Food and Drugs Act* of 1908-1935, of South Australia.

* * *

Dr. L. W. Jeffries, Dr. C. T. Ch. de Crespigny and Dr. W. Ray have been appointed Members of the Council of the Institute of Medical and Veterinary Science, under the provisions of the *Institute of Medical and Veterinary Science Act*, 1937, of South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi to xviii.

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Clinical Assistants.
BROUGHTON HALL PSYCHIATRIC CLINIC, SYDNEY, NEW SOUTH WALES: Honorary Out-Patient Psychiatrist.
GOVERNMENT OF FIJI: Medical Officer.
ROYAL NEW ZEALAND SOCIETY FOR THE HEALTH OF WOMEN AND CHILDREN, DUNEDIN, NEW ZEALAND: Medical Adviser.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 175, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.

AUG 3 1938

Registered at the G.P.O., Sydney, for Transmission by Post as a Newspaper. Published Weekly. Price 1s.

MEDICAL LIBRARY

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—25TH YEAR. SYDNEY, SATURDAY, JUNE 25, 1938. No. 26.

"PLOUGH" BRAND
EXTRACT OF MALT & COD-LIVER OIL

The Cod-Liver Oil in this fine preparation is of the highest grade, freshly prepared in our own factories which are located as near as possible to the fishing grounds where the Cod are caught. This Oil is rich in Vitamins "A" and "D", as determined by the most approved Biological and Chemical tests. The Oil is finely emulsified with an extract made from the best Barley Malt and the emulsion is suitably flavoured with aromatics and carminatives, thus producing a rich, easily digestible and palatable compound.

"Plough" Brand Extract of Malt & Cod-Liver Oil is a most valuable accessory nutrient for people of all ages. It is used with great benefit for improving nutrition in a variety of diseases and in convalescence. It is an admirable preparation for children, especially those who are sickly or under-developed and have a tendency to rickets and a low resistance against infection. It is readily taken by children on account of its pleasant taste, and is not liable to cause any digestive trouble.

Prices: 1 lb. jars, 2/6; 2 lb. jars, 4/6.

ALLEN & HANBURYS (AUSTRALASIA) LTD.

41, Hunter Street, SYDNEY.

Duo-Therapy sunshine for *your* patients—

Simultaneous (general) tonic irradiation with ultra-violet rays from the Alpine Sun Lamp and (regional) lumbar infra-red irradiation with the Sollux Lamp illustrating the technique for treating lumbago.

There is no reason why you should go without the benefits which actino-therapy brings to both physician and patient. But your results depend largely on effective apparatus. No skill or study will enable you to overcome self-imposed defects due to inadequate or outworn equipment.

Specify the Hanovia Duo-Therapy Unit and have a lamp which is always dependable, always ready for use, always constant in its high intensity of ultra-violet radiation. Remember that this Unit combines a modern ultra-violet ray lamp (the self-starting electronic Alpine Sun) with the standard therapeutic infra-red lamp (the new Sollux, Model V). Every model embodies the manufacturers' specialised experience of 32 years in making sunlamps. That is why the Duo-Therapy Unit enables you to supply any group and combination of rays needed for modern actinotherapy, in full therapeutic intensity, following the best technique, and using a dependable, responsive instrument. For hospital, clinic or consulting room, you will find the Duo-Therapy Unit satisfies all your requirements as it has satisfied thousands of your colleagues.

We offer Hanovia's full service for information, installation, and technical assistance to enquirers, and their comprehensive guaranty, Technique Handbook and special actino-therapy magazine to professional users.

The coupon is for your convenience

WATSON VICTOR LIMITED

Watson House, 9-13 Bligh Street, Sydney

MELBOURNE, BRISBANE, PERTH, ADELAIDE, NEWCASTLE

TO WATSON VICTOR LTD.,
Watson House, Bligh Street, Sydney.

Dear Sirs,

Please send me handbooks on ultra-violet and infra-red irradiation with the Hanovia Duo-Therapy Unit.

Name .....

Address .....

M63/70 .....

TONICS APLENTY—BUT . . .

Of tonics there is a seemingly endless variety, but WATERBURY'S COMPOUND is the tonic for convalescents. Cod liver oil, fresh liver, spleen, pancreas, predigested and made easily assimilable, with the hypophosphites of calcium and potassium, and unfermented extract of malt, is what the depleted organism needs.

The properties of WATERBURY'S COMPOUND PLAIN are intended for reconstructive purposes, but there is WATERBURY'S COMPOUND with Creosote and Guaiacol for those convalescents whose recovery is hindered by stubborn cough.

A test will demonstrate their therapeutic value. We shall be glad to send trial quantities.

WILLIAM R. WARNER & CO. LTD.,
520, Riley Street, Sydney, N.S.W.

Made by Waterbury Chemical Co., Inc.—New York & St. Louis, U.S.A.

Jenyns Surgical Corsetry

for effective control and support

Correct for post-operative wear and in all cases of visceroptosis or general weakness.

We specialise in making and fitting surgical Belts, Corsets, Girdles, Corselettes, Brassieres, Spinal Supports, Trusses and all kinds of Elastic Supports.

● *Prices for special garments and further information will readily be supplied upon request.*

THE JENYNS PATENT CORSET PTY.
309-315 George Street, Brisbane

MADE IN AUSTRALIA

COMMONWEALTH OF AUSTRALIA

Department of Health

Commonwealth "INSULIN"

Insulin is available at the following prices:

5 c.c. phial of 100 units (20 units per c.c.)	1/6
5 c.c. phial of 200 units (40 units per c.c.)	2/10
5 c.c. phial of 300 units (60 units per c.c.)	4/3

Every batch of Commonwealth Insulin is carefully tested before issue and the UNIT EMPLOYED IS THAT OF THE STANDARD ISSUED UNDER THE AUTHORITY OF THE LEAGUE OF NATIONS.

COMMONWEALTH INSULIN IS — HIGHLY PURIFIED, STABLE, ACTIVE, STERILE.

Commonwealth PITUITARY EXTRACT

IS PHYSIOLOGICALLY STANDARDIZED IN ACCORDANCE WITH THE RECOMMENDATIONS OF THE LEAGUE OF NATIONS TECHNICAL CONFERENCE.

1 box of 6 × 1 c.c. Surgical, 20 International Units per c.c.	8/6
1 box of 6 × ½ c.c. Surgical, 20 International Units per c.c.	5/-
1 box of 6 × 1 c.c. Obstetrical, 10 International Units per c.c.	5/-
1 box of 6 × ½ c.c. Obstetrical, 10 International Units per c.c.	3/6

Supplies may be obtained from the Commonwealth Serum Laboratories, Parkville, Victoria, and also from the following: Director-General of Health, Canberra; Chief Quarantine Officer (General), Anzac Square, Adelaide Street, Brisbane, Qld.; Chief Quarantine Officer (General), Customs House, Circular Quay, Sydney, N.S.W.; Chief Quarantine Officer (General), C.M.L. Building, 41-47 King William Street, Adelaide, S.A.; Chief Quarantine Officer (General), G.P.O., Perth, W.A.; Chief Quarantine Officer (General), Commonwealth Health Laboratory, Launceston, Tasmania.

COMMONWEALTH SERUM LABORATORIES

PARKVILLE, N.2 — VICTORIA — AUSTRALIA

Wholly set up, printed and published at The Printing House, Seamer Street, Glebe, Sydney, N.S.W., by ARTHUR FREDERICK ROOTS SIMPSON, of Fairholm Street, Strathfield, New South Wales, on behalf of the AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, Seamer Street, Glebe, Sydney, New South Wales.